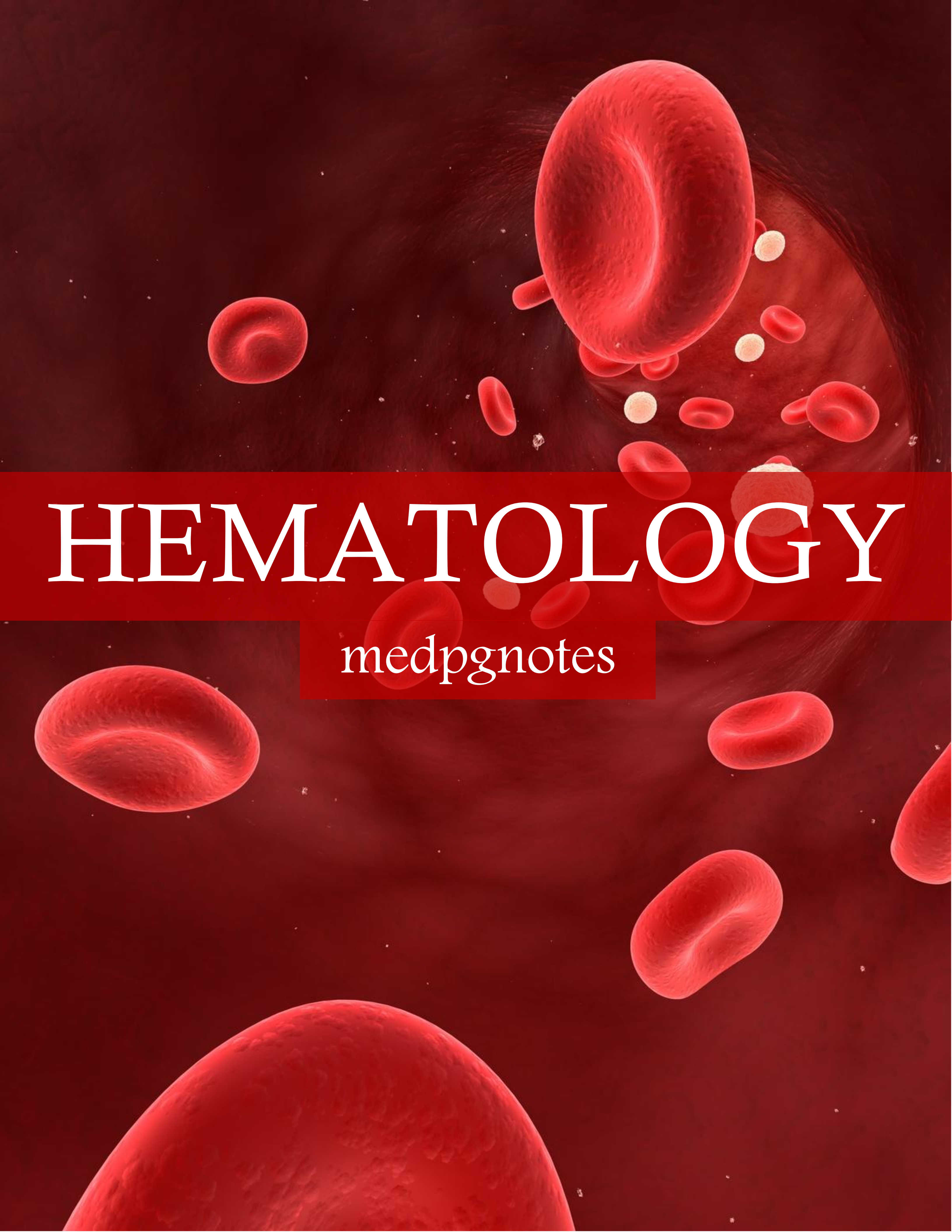

HEMATOLOGY

medpgnotes

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KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two.

RED BLOOD CELLS

GENERAL FEATURES OF RED BLOOD CELLS

Most abundant protein in RBC membrane	**Glycophorin (Band 3)**
NOT a RBC membrane protein	Nebulin
Biconcave shape of RBC helps by	Passing easily through smaller capillaries, Allows considerable alteration in cell volume, Resist hemolysis
Transport in RBC is	**Linear shaped**
Thrombosthenin is	Contractile protein
Thrombospondin	Angiogenesis inhibitory protein
Protein that contributes maximum for ESR	Fibrinogen
Lizenmeyer is used to measure	ESR
ESR is zero in	Afibrinogenemia

RED CELL INDICATORS

$MCV =$	$\frac{Hematocrit}{RBC\ count} * 10$
Normal MCV	86 - 98 micrometre cube
Normal MCH	28 - 33 g/dl
Normal red cell distribution width	Less than 14.5%
Serum iron	50 - 150 microgram/dl
Serum total iron binding capacity	300 - 600 microgram/dl
Hematocrit is more in venous blood due to	*Chloride shift*

ERYTHROPOIETIN

NOT a transport of binding protein	Erythropoietin
Site of production of erythropoietin	*Peritubular capillaries*
Erythropoietin is produced in	Kidney
Erythropoietin is associated with	Increased reticulocytes, increased hematocrit, increased hemoglobulin, eliminates need for transfusion
NOT true about erythropoietin	Decrease in reticulocyte count
Erythropoietin levels are increased by	Decreased tissue pO2 concentration
Erythropoietin is NOT increased in	Pancreatic carcinoma
Low erythropoietin level	Renal failure
Erythropoietin is NOT secreted by	Adrenocortical tumor
Erythropoiesis is NOT promoted by	Estrogen
Erythropoietin is used in	Chronic renal failure, Carcinoma chemotherapy, Myelodysplastic syndrome

HEMATOPOIESIS

Fetal hematopoiesis first occurs in	Yolk sac
Fetal erythropoiesis first occur at	6th week of gestation
During first 3-4 month of gestation, erythrocytes are formed from	Yolk sac, Liver, Spleen
Major site of active hematopoiesis in a fetus of 5 months	Liver
Hematopoiesis from 5th to 9th month of gestational age	*Bone marrow*
Site of RBC formation in 20 year old healthy male	Flat bones
Most Potent stimuli for Erythropoiesis	Hypoxia
Normal hematopoiesis does not require	Calcium
Drug used to release stem cells from bone marrow	**Plerixafor**

PERIPHERAL BLOOD SMEAR

Fragmented RBC's in peripheral blood	Microangiopathic hemolytic anemia, DIC, Malignant hypertension, HELLP syndrome, sepsis,HUS, eclampsia, scleroderma
Nucleated RBCs	Erythroblastosis
Reticulocyte count increased in	Treated iron deficiency anemia, Myeloid metaplasia, sideroblastic anemia
Reticulocytosis is NOT seen in	Anemia in chronic renal failure, Nutritional anemia, congenital dyserythropoietic syndrome
Extreme microcytosis is associated with	*Hereditary pyropoikilocytosis*
Macrocytes in blood smear	Liver disease, Aplastic anemia, Vitamin B12 and folate deficiency
Spherocytes in peripheral smear	Hereditary spherocytosis, Hemoglobin C
Microspherocytosis	ABO incompatibility
Echinocytes are type of	RBC
Burr cells	Uremia
Spur cells	**Laennec cirrhosis**
Spur cell	**Acanthocytosis**
Acanthocytes	Abetalipoproteinemia
Somatocytes	**Central pallor**
Stomatocytes	**Slit in RBC**
Xerocytosis	**Dehydrated red cells**
Schistocyte	**Helmet shaped**
Schistocytes are not seen in	*March hemoblobinuria*
Dacryocyte	**Tear drop shaped**
Echinocyte	**Spiculated**
Acanthocyte	**Irregular spike**
Poikilocyte	**All abnormal cells**
Degmacyte	*Bite cell*
Drepanocyte	*Sickle cell*
Sideroblasts are seen in	Myelofibrosis, alcoholism, iron overload
Pappenheimer bodies are seen in	*Severe anemia, thalassemia*

Pappenheimer bodies composed of	Iron
Gamma gandy bodies contain	Hemosiderin and calcium
Gamma gandy nodules	Spleen
Gamma gandy bodies are seen	CML, Long standing thalassemia, Cirrhosis of liver
Seen on Romanowsky stain	Basophilic stippling, Howell Jolly bodies, Cabot ring
Basophilic stippling	**5' nucleotidase deficiency**
Polychromatophilia	**Large cells with light purple colouring**
Pelger heut anomaly	**Bilobed neutrophils, spectacle like or pince nez configuration**

HEMOGLOBIN

Embryonic hemoglobin	*Gower 1 (zeta 2, epsilon 2), Gower 2 (alpha 2, epsilon 2), Portland (zeta 2, gamma 2)*
Fetal hemoglobin	*HbF (alpha 2, gamma 2)*
Adult hemoglobin	*HbA1 (alpha 2, beta 2), HbA2 (alpha 2, delta 2)*
Gower hemoglobin in newborn is associated with	*Trisomy 13*
Hb Bart	*Gamma 4 (alpha thalassemia)*
HbH	*Beta 4 (alpha thalassemia)*
Most unstable hemoglobin in children	*Hb Kob*
Hemoglobin appearing first in fetus	Hb gowers
Proportion of HbF at birth of term infant	75%
Switchover from fetal to adult hemoglobin begins	36 weeks of gestation
Fetal Hb is replaced completely by adult hemoglobin by	6 months
% of HbF in 6 months old infant	10%
Fetal Hb	Resistant to alkali denaturation
30 year old female asymptomatic, not requiring blood transfusion has Hb 13 gm%, HbF 95%, HbA2 1.5%. Diagnosis	Persistently raised HbF
Hb bart is lethal because	Hb bart cannot release oxygen to fetal tissues
Increased fetal Hb seen in	Juvenile CML, Hereditary spherocytes
Hemoglobin F is raised in	Juvenile CML
Iron in hemoglobin in	**Ferrous form**
1 heme carry	**1 02**
1 hemoglobin can carry	**Up to 4 oxygen**
Salt bridges are seen in	**Deoxyhemoglobin**
Abnormal hemoglobin associated with polycythemia	**Yakima**
Hb Yakima	*High affinity*
Hb Kansas	*Low affiniity*
Hb Itawa	**Methemoglobinemia**
Muddy appearance of blood	*methemoglobinemia*
Congenital methemoglobinemia is associated with	*NADP diaphorase deficiency*
HbA2 levels increased in	Beta thalassemia, sickle cell anemia, megaloblastic anemia, hyperthyroidism
HbH is associated with	Deletion of 3 alpha genes
Erythroleukemia is associated with	**HbH disease**

What is affected in HbS	Solubility
NOT true about hemoglobin S	Binding of HbS to deoxygenated HbA can extend the polymer and cause sickling of red blood cells
HbE is common in	West Bengal
Quantity of hemoglobin released everyday in a normal adult	8 gram
Hemoglobin estimation is NOT done by	Wintrobe
Isopropanolol stability test is for	*Unstable hemoglobin*

GENERAL FEATURES OF RED BLOOD CELL DISORDERS

Iron overload occurs in	Thalassemia, myelodysplastic syndrome, sideroblastic anemia
Bone marrow iron is increased in	Sideroblastic anemia, Anemia of chronic disease, Megaloblastic anemia, Pernicious anemia, Thalassemia
Microcytosis is seen in	Thalassemia, Hb Lepore, Hb Bart
Decreased osmotic fragility	Sickle cell disease,Thalassemia
Low ESR	Sickle cell anemia, hereditary spherocytosis, hereditary elliptocytosis
Pancytopenia with hypercellular marrow	Megaloblastic anemia>PNH
Pancytopenia with hypercellular marrow is NOT a feature of	Dyskeratosis congenita
Fanconi anemia	Constitutional anemia
Inheritance of Fanconi anemia	*Autosomal recessive*
Congenital hypoplastic anemia (diamond blackfan syndrome)	*Anemia, reticulocytopenia, decreased RBC precursors in bone marrow*
Congenital dyserythropoietic anemia type II	*RBC lysis in acid fast serum from normal persons but NOT in patient's serum*
Massive blood loss in normal patient	Increased MCV, Thrombocytosis, Reticulocytosis
37 year multipara has blood picture showing hypochromic anisocytosis	Combined iron and folic acid deficiency
Spur cell anemia is caused by	Chronic liver disease

HYPOCHROMIC MICROCYTIC ANEMIA

Microcytic hypochromic anemia	Thalassemia, iron deficiency anemia, sideroblastic anemia, lead poisoning
MC cause of microcytic hypochromic anemia in India	Hookworm infection
Anemia caused by pulmonary TB	Microcytic hypochromic anemia
Iron deficiency anemia leads to	Microcytic hypochromic anemia
Microscopic hypochromic anemia is seen in	Sideroblastic anemia
Copper deficiency associated with	Microcytic hypochromic anemia
Hypochromic Microcytic anemia is NOT associated with	Sickle cell anemia
Microcytic anemia NOT caused by	Fanconi's anemia
Hypochromic anemia is NOT seen in	Fanconi anemia
Microcytic hypochromic anemia with normal level of free erythrocyte protoporphyrin	Thalassemia
Pathogenesis of hypochromic anemia in lead poisoning	Inhibition of enzymes involved in heme synthesis

is	
Nonspecific symptoms of anemia, target cells. hypochromic microcytic anemia. positive family history	Hb electrophoresis

IRON DEFICIENCY ANEMIA

Low iron causes	*Increased transferrin mRNA stability*
Iron absorption is increased in	Iron deficiency, pregnancy, hypoxia, acidic pH, ferrous iron salts
Malnutrition in India attributed to	Iron deficiency protein deficiency, vitamin A deficiency
NOT a cause of iron deficiency anemia	Chronic renal failure
Stages in iron deficiency	**Negative iron balance, iron deficiency erythropoiesis, iron deficiency anemia**
Earliest change of iron deficiency anemia	Decreased serum ferritin
Most sensitive indicator of Iron deficiency anemia	Serum ferritin
Most suitable test to asses iron stores	Serum ferritin
First indices to change in iron deficiency anemia	Serum ferritin
Ferritin stores are NOT present in	Bone
Ferritin is NOT seen in	Spleen
Iron deficiency anemia	Increased free erythrocyte Protoporphyrin
Iron deficiency anemia	Increased TIBC, decreased serum ferritin, Bone marrow iron decreased earlier than serum iron
In iron deficiency anemia	Red cell distribution width increases
Transferrin has	**2 iron binding sites**
Best test to detect iron deficiency in community	Serum transferrin
Glycoprotein transported in plasma iron metabolism	Transferrin
Transferrin saturation	**Serum iron * (100/TIBC)**
Normal transferrin saturation	**30 – 50 %**
Indirect measure of transferrin	**TIBC**
NOT true regarding iron deficiency anemia	Transferrin saturation < 16%
Best test with respect to highest positive predictive value and specificity to rule out iron deficiency	*Bone marrow stain for iron*
NOT true about anemia	Treatment should be discontinued immediately once hemoglobin normalizes to prevent side effects of iron
Macrocytic anemia NOT seen in	Iron deficiency
NOT seen in iron deficiency anemia	Hypersegmented neutrophils
Oral therapy for iron deficiency anemia	Decrease in absorption with absorption with symptoms
Iron requirement	4*wt in kg*Hb deficit in gm/dl
If 200 – 300 mg of elemental iron consumed	50 mg absorbed
Percentage of elemental iron present in hydrated ferrous sulphate	33%
Iron supplementation in a healthy term breast fed infant should be started at the age of	6 weeks
Response to iron therapy in iron deficiency anemia	Reticulocytosis
Reticulocytosis after iron therapy in Iron deficiency anemia seen after	4 – 7 days
First sign of improvement noted after iron therapy is initiated	*Decreased irritability*

For oral iron supplements used for iron deficiency anemia	Tolerable dose will deliver 40 – 60 mg of iron per day
Indication for intramuscular iron therapy	Oral intolerance

MEGALOBLASTIC ANEMIA

TC II deficiency can cause	*Megaloblastic anemia (but TC I deficiency does not cause megaloblastic anemia)*
Babies fed exclusively on goat milk develop	Megaloblastic anemia
Megaloblastic anemia in blind loop syndrome due to	Bacterial overgrowth
Megaloblastic anemia due to folic acid deficiency is commonly due to	Inadequate dietary intake
Ileocaecal tuberculosis is associated with	Megaloblastic anemia
Megaloblastic anemia is a side effect of	Phenobarbitone,Phenytoin,Methotrexate,Pyrimethamine,Phenytoin,sulfasalazine,alcohol
Macrocytic anemia may be seen with	Liver disease, thiamine deficiency, orotic aciduria
Macrocytic anemia	Vitamin B12 deficiency, Zidovudine therapy, Myelodysplasia
Anemia associated with raised MCV and normal MCHC	Vitamin B12 and Folic acid deficiency
Causes of vitamin B12 deficiency megaloblastic anemia	Fish tape worm infestation, Gastrectomy, Ileal resection
Causes of folate deficiency megaloblastic anemia	Phenytoin therapy, Methotrexate
Macrocytic anemia	Hypothyroidism, Orotic aciduria, Lesch Nyhan syndrome
Megaloblastic anemia develop in	Children fed on goat milk
Megaloblastic anemia	Alcohol, Phenytoin, Sulfasalazine
Deficiency of folic acid produce	Megaloblastic anemia
Drug causing megaloblastic anemia	Pyrimethamine
NOT a cause of Macrocytic anemia	Lead poisoning
NOT a cause of macrocytic anemia	Pentamidine
Macrocytic anemia NOT associated with	Ciprofloxacin
NOT a cause of megaloblastic anemia	Choloroquine
NOT a cause of megaloblastic anemia	Aspirin
NOT a cause of macrocytic anemia	*Abetalipoproteinemia*
Childhood anemia with increased pigmentation of back and fingers	*Megaloblastic anemia*
Earliest Hematologic abnormality of Megaloblastic Anemia	Hypersegmented Polymorphs
Megaloblastic anemia associated with	Increased LDH
Anemia associated with pancytopenia	Megaloblastic anemia
Macropolycytes	Megaloblastic anemia
Maturation failure in poor absorption of vitamin B12 is associated with	Anemia occurs after 3-4 months of exposure
Metabolism of FIGLU is impaired in	Folate deficiency
False regarding megaloblastic anemia	Reticulocyte count increased
Macroglossia, Hb 11.5, MCV 100,next step	B12 estimation
Moderate anemia, MCV 45, Diagnosis by	Vitamin B12
Shilling test is used to determine deficiency of	Vitamin B12
Macrocytosis in complete blood count diagnosed by	Increased MCV
Folic acid deficiency best determined by	Increased FIGLU in urine
Patient on phenytoin, develops depression, prescribed	MCV should be estimated

tricyclics, complaints of lassitude and his Hb 8. Next step	
In treatment of megaloblastic anemia, vitamin B12 and folic acid should be given together because	Folic acid alone causes improvement of anemic symptoms but neurological dysfunction continues
Megaloblastic anemia secondary to folate deficicency corrected by administering folate	2 mg daily
B12 and Folic acid correct megaloblastic anemia by	Increasing DNA synthesis

PERNICIOUS ANEMIA

Intrinsic factor of castle is secreted by	Parietal cells
Deficiency of intrinsic factor of Castle cause	Pernicious anemia
Pernicious anemia	Polycytosis, atrophy of gastric mucosa, increased urobilinogen in urine
Moeller's glossitis	Pernicious anemia
Hypogastrinemia with hypochlorhydria	Pernicious anemia
Raised gastrin secretion without increase in Hcl secretion	Pernicious anemia
Raised gastrin level without associated increase in acid secretion	Pernicious anemia
Carcinoma of stomach develops secondarily to pernicious anemia in	Fundus
MC Site of Gastric Adenocarcinoma secondary to Pernicious Anemia	Fundus & Pylorus
NOT true regarding pernicious anemia	Antibodies to Vitamin B12
Serum gastrin in pernicious anemia	Elevated

ANEMIA OF CHRONIC DISEASE

Anemia in chronic renal failure is due to	Decreased erythropoietin production
Anemia of chronic renal failure	Normocytic normochromic anemia, erythropoietin improves symptom, anemia is proportional to kidney disease
Hypoproliferative anemia associated with decreased serum ferritin with normal serum iron. these features are seen in	Chronic Inflammation
Anemia of chronic disease	Normal or increased serum ferritin
Seen in chronic inflammatory anemia	Serum iron decreased, serum ferritin increased, serum transferrin decreased
Anemia of chronic disease	Decreased serum iron, decreased TIBC, increased ferritin, increased bone marrow iron
Anemia of chronic disease is characterized by	Increased Sideroblasts, Decreased serum iron, serum total iron binding capacity
Anemia of Chronic disease may be both	Normocytic/Microcytic
Serum iron and serum TIBC decreased in	Anemia of chronic disease
Anemia of chronic disease differentiated from iron deficiency anemia by	Decreased TIBC, increased ferritin
Macrocytic anemia NOT seen with	Anemia of chronic disease
Epoietein alpha is used in treatment of	Anemia of chronic disease

SIDEROBLASTIC ANEMIA

Metal causing sideroblastic anemia	Lead
33 yr Alcoholic, increased serum iron and increased serum transferrin	Sideroblastic anemia
Elevated serum ferritin with normal iron stores	Chronic disease, sideroblastic anemia
Increased serum ferritin, decreased TIBC, increased serum iron percentage saturation increased	Sideroblastic anemia

FEATURES OF HEMOLYTIC ANEMIA

Most common glycolytic pathway defect leading to hemolytic anemia	*Pyruvate kinase deficiency*
Hemoglobinuria	Mismatched blood transfusion, paroxysmal cold hemoglobinuria, thermal burns, snake bite, CuSO4 poisoning
Haptoglobin levels are decreased in	Mismatched blood transfusion, thalassemia, G6PD
Direct Coombs test in hemolytic anemia	Positive
Positive coomb test associated with	Methyldopa
Direct coomb's test detects	Antibodies in serum
Direct Coombs test is positive in hemolytic anemia due to	Paroxysmal cold Hemoglobinuria
Coomb's positive hemolytic anemia	Chronic active hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis
Coomb's positive hemolytic anemia	HUS,PAN,SLE
Coomb's positive hemolytic anemia	SLE, scleroderma, PAN
Cause mild to severe Coomb's positive hemolytic anemia	Atypical mycobacteria
Coomb's positive hemolytic anemia is NOT seen in	Alcoholic cirrhosis
Methyldopa	Hemolytic anemia in pregnant women
Acute intravascular hemolysis is associated with	Bartonella bacilliformis, plasmodium falciparum, Babesi microti
Intravascular hemolysis	Mismatched blood transfusion, snake bite, PNH, Acute G6PD deficiency
First sign of Hemolysis	Decreased Haptoglobin
In hemolytic anemia	Diploic space WIDENS
Features of hemolytic anemia	Decreased RBC life span, decreased haptoglobin, unconjugated hyperbilirubinemia, altered erythroid and myeloid ratio, hemoglobinuria, reticulocytosis, hemosiderinuria
Features of hemolytic anemia	*Increased carboxyhemoglobin, increased LDH, increased plasma hemoglobin, decreased hemoglobin and decreased hemopexin*
Cold hemolytic anemia is associated with	*Mycoplasma and infectious mononucleosus*
Hemolysis is associated with	**Increased LDH**

Reticulocyte count in Hemolytic anemia	Increased
NOT true about Hemolytic anemia	Spherocytosis
NOT seen in hemolytic anemia	Increased haptoglobin
NOT seen in hemolytic anemia	Tear drop and burr cells
NOT associated with severe hemolysis	Clostridium TETANI

HEREDITARY SPHEROCYTOSIS

Aplastic anemia in hereditary spherocytosis is precipitated by	Parvo virus
Hereditary spherocytosis can be caused by defects in	Ankyrin, Palladin, Anion transport protein
Hemolytic anemia due to intrinsic cell defect	Hereditary spherocytosis
Autosomal recessive type of hereditary spherocytosis is associated with	*Mutation in alpha spectrin band 42*
Intracorpuscular Defect of Erythrocyte	Hereditary Spherocytosis
MC mutation in Hereditary Spherocytosis	Ankyrin
Least Common defect in Hereditary Spherocytosis	Palladin
Severe form of hereditary spherocytosis	Alpha spectrin
Increased osmotic fragility	Hereditary ellipsocytosis
Almost only one condition where MCHC is high is	Hereditary Spherocytosis
Hereditary spherocytosis	MCV decreased, MCHC increased, osmotic fragility increased
NOT associated with hereditary spherocytosis	*Glycophorin C*
Autohemolysis test is positive for	Hereditary spherocytosis
Pink test for	**Hereditary spherocytosis**
Important test to confirm Hereditary Spherocytosis	Osmotic fragility
Coomb's test in hereditary spherocytosis	Negative
Coomb's test	Antiglobin test
Spherocytes in hereditary spherocytes show	Smaller size, Reticulocytosis seen, Increased MCHC
Investigation to be done if peripheral smear shows spherocytes	Coomb's test
Hereditary Spherocytosis is best treated with	Splenectomy
Splenectomy is most useful in	Hereditary spherocytosis
Optimum time for splenectomy in child with Hereditary spherocytosis	3-4 years of age

G6PD DEFICIENCY

Inheritance of G6PD Deficiency	X-linked recessive
G6Pd deficiency is common in	Males
Hemolysis in presence of Oxidative stress of some drugs	G6PD deficiency
Drug contraindicated in G6PD deficiency	Cotrimazole, Nitrofurantoin, Primaquine
Contraindicated in G6PD deficiency	Cotrimoxazole, furazolidone, nalidixic acid
In G6PD deficiency, hemolysis occurs commonly with	Primaquine
NOT causing hemolysis in G6PD	Pyrimethamine
Heinz Bodies	G6PD deficiency
Heinz bodies are precipitates of	Globin

Bite Cells (hemighost, blister cells)	G6PD Deficiency

SICKLE CELL ANEMIA

Sickle cell anemia	Autosomal recessive
Mother has sickle cell disease, father is normal, chances of children having sickle cell disease and sickle cell trait respectively	0 and 100%
Sickle cell disease	Glutamine to valine, RFLP results from single base change, HbS confers resistance against malaria in heterozygotes
Defect in sickle cell anemia in	Beta chain
Defect in sickle cell anemia	Base substitution in DNA
Primary defect in sickle cell anemia	Replacement of glutamate by valine in beta chain of HbA
Can be a homologous substitution for valine in hemoglobin	Glutamic acid
Event responsible for mutation in B chain in sickle cell anemia	Point mutation
HbS has defect in aminoacid position	6
Defect in hemoglobin of sickle cell anemia is due to replacement of	B6 Glutamate by Valine
Replacement in sickle cell anemia	Glu by val in B6
In a mutation, if valine is replaced by which of the following will NOT result in any change in function of protein	Aspartic acid
Sickling in HbS is mainly caused by	Decreased solubility
Factors aggravating sickling	Higher concentration of HbS, lower concentration of HbC, fall in blood pH
American male with recurrent abdominal pain, chronic mild hemolysis, abnormal shaped RBC. Long standing non healing leg ulcer. Defect in	Single nucleotide replacement
MC presentation of Sickle cell anemia	Bone pain
Most Dangerous crisis in Sickle cell Anemia	Sequestration crisis
In sickle cell anemia, sudden onset of thrombocytopenia with hemolysis and two of reticulocyte count occurs in	Sequestration crisis
Modality to be avoided in sickle cell anemia	IV regional anaesthesia
Which does not cause increase in size of Spleen in Late stage	Sickle Cell anemia
Salmonella Osteomyelitis is seen in	Sickle cell anemia
Heterozygous sickle cell anemia gives protection against	Malaria
Sickle cell anemia leads to resistance against	Plasmodium falciparum
Sickle cell trait patient do not have manifestations as that of sickle cell disease	50% HbS is required for occurrence of sickling
Radiological features of sickle anemia	Vertebra plana, Bone infarct, Marrow hyperplasia, Secondary osteomyelitis
Fish Mouth Vertebra	Sickle cell anemia
Autosplenectomy	Sickle cell anemia
Hair on end Skull (wide diploic space of skull with brush	Thalassemia, Sickle Cell Anemia

border)	
Skull X ray of Sickle cell anemia	Crew cut
HbS in electrophoresis is associated with	*Slow motility*
Mandatory vaccine in HbS	*Pneumococcal vaccine*
NOT true about sickle cell anemia	Increased ESR
NOT an autosomal disease	Sickle cell anemia
Sequestration crisis of sickle cell anemia is NOT characterized by	Reduced platelets
NOT seen in chronic case of sickle cell anemia	Splenomegaly
NOT true about sickle cell disease	Sickle patch is generated as a result of replacement of non polar residue with a polar residue
NOT true about sickle anemia	Leukopenia
NOT true about sickle cell disease	Patient presents early in life before 6 months of age
NOT true about sickle cell disease	Mutation in alpha chain
NOT true about sickle cell disease	Deoxygenated Hb leading to exposure of sticky end due to replacement of non polar residue by polar residue
Methods used to detect point mutation in beta globulin gene that causes sickle cell anemia	Polymerase chain reaction with allele specific oligonucleotide hybridization, southern blot analysis, DNA sequencing
Seen on hemoglobin electrophoresis in sickle cell anemia	HbA2,HbF,HbS
NOT a method used to detect point mutation in sickle cell anemia	Northern blot
Drug used in sickle cell anemia	Hydroxyurea
Methotrexate is NOT used for	Sickle cell anemia

THALASSEMIA

MC Type of Hemoglobinopathy in World	Thalassemia
MC Type of mutation causing β-Thalassemia	Splicing mutation
MC Site of Mutation in Thalassemia	Intron I (Intervening sequence)
MC gene mutation in beta thalassemia	Intron 1 inversion
Most thalassemia are consequences of mutation causing	*RNA processing or production defects*
Thalassemia occurs due to	Splicing mutation, Frame shift mutation
MC mutation in Beta Thalassemia	Point Mutation
Allelic Heterogenecity	Beta-Thalassemia
Risk of beta thalassemia with thalassemia in distant relative	0%
In β-Thalassemia	β-chains are deficient
Osmotic Fragility in Beta Thalassemia	Decreased
Thalassemia gives protection against	Malaria
Chipmunk facies	**Beta thalassemia**
Hair on end appearance	Thalassemia
25 year female presented with mild pallor, moderate hepatosplenomegaly, Hb 9.2 g/dl, fetal hemoglobin 65%,NOT received any blood transfusion till date	Thalassemia intermedia
23 year female asymptomatic MCV 70,ferritin 100 g/L, hb 10 gm%	Thalassemia trait

Diagnostic findings in Beta Thalassemia trait	Increased HbA2, Increased HbF, Microcytosis
Beta thalassemia trait	Increased HbF, Increased HbA2, Microcytosis, Target cell
Target cell	**Codocytes**
Microcytic anemia MCV with normal total RBC count	Thalassemia minor
Most dangerous form of Alpha thalassemia	Hydrops fetalis
Alpha thalassemia is associated with	*Decreased HbA1, decreased HbA2, decreased HbF*
Alpha thalassemia trait is associated with	Microcytosis
Child died soon after birth, hepatosplenomegaly and edema all over the body	Alpha thalassemia
Dactylitis NOT seen in	Beta thalassemia
NOT true beta thalassemia trait	Patient require blood transfusion, Severe anemia
NOT true about beta thalassemia	Increased osmotic fragility
NESTROFT test is for	Beta thalassemia
Investigation of choice for Thalassemia	Hemoglobin Electrophoresis
Investigation needed to estimate Thalassemia trait	HbA2 Electrophoresis
Hemoglobin estimation helps in diagnosis of beta thalassemia trait	HbA2
Treatment of choice for thalassemia major	Iron, blood transfusion, desferrioxamine
Most appropriate drug for chelation in beta thalassemia major	Oral deferiprone
Level of Hemoglobin required for maintaining good body growth and normal activities in beta thalassemia patient	*10 – 12 gm%*

AUTOIMMUNE HEMOLYTIC ANEMIA

Warm autoimmune hemolytic anemia seen in	SLE, alpha methyl dopa therapy, Non Hodgkin's lymphoma
NOT warm autoimmune hemolytic anemia	Mycoplasma pneumonia
Cold antibody immune hemolytic anemia is associated with	Mycoplasma, Infectious mononucleosis, Paroxysmal cold hemoglobinuria
21 year male, anemia, mild hepatosplenomegaly, hb 5 mg%. history of single blood transfusion	Autoimmune hemolytic anemia
Autoimmune hemolytic anemia associated with malignancy of	B cell lineage
Autoimmune hemolytic anemia	CLL
Spherocytes in peripheral smear	Autoimmune hemolytic anemia
NOT an absolute indication of splenectomy	Autoimmune hemolytic anemia

MICROANGIOPATHIC HEMOLYTIC ANEMIA

Examples of Microangiopathic Hemolytic anemia	TTP, HUS, DIC, microscopic polyangitis, prosthetic valve
Microangiopathic hemolytic anemia NOT seen in	APLA, ITP

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

Stem cell disorder affecting all three lines RBC, WBC and platelets	Paroxysmal nocturnal hemoglobinuria
Paroxysmal Nocturnal Hemoglobinuria is basically due to	Acquired Red cell defect
PNH	Only hemolytic anemia caused by acquired intrinsic defect
PNH is associated with	Aplastic anemia, venous thrombosis, iron deficiency anemia
PNH associated with	Cerebral thrombosis, Budd chiari syndrome, pancytopenia, hemosiderinuria, cellular marrow
Paroxysmal nocturnal hemoglobinuria	**Complement mediated destruction of CD59 red cells, shortage of GPI due to mutation in PIG-A gene**
PNH is associated with	*Red cell lysis in patients's own acidified serum*
Deficient protein in Paroxysmal nocturnal hemoglobinemia	CD8,CD3b (CD 55)
PNH is mainly due to deficiency of	CD59
PNH is associated with deficiency of	DAF, MIRL, GPI anchored protein
PNH can lead to	*AML*
Congenital Abnormality is NOT seen in	Paroxysmal Nocturnal Hemoglobinuria
Pancytopenia with cellular marrow	PNH
PNH is associated with somatic mutation affecting	Glycosylated phosphatidyl Inositol
5 year old male child with episodic anemia and jaundice, he is LEAST likely to have	Paroxysmal nocturnal hemoglobinuria
Leading cause of death in PNH	Thrombosis
NOT seen in PNH	Arterial thrombus
NOT associated with PNH	Massive splenomegaly
Gold standard test for diagnosis of PNH	Flow cytometry
Leucocyte Alkaline Phosphatase DECREASED in	PNH, CML
PNH associated with	Increased LDH, Decreased LAP, Increased hemosiderin in urine
Screening test for PNH	*Sucrose hemolysis test (sugarwater test)*
Confirmatory test for PNH	*Ham test*
Ham test is based upon	Complement
Ham test is associated with	Defective GPI anchor
Treatment of PNH	**Eculizumab (blocks C5 so that MAC is not formed)**
Rituximab NOT used for	PNH
Splenectomy for PNH	Not useful

PAROXYSMAL COLD HEMOGLOBINURIA

Paroxysmal cold hemoglobinuria is triggered by	**Viral infection**
Stem cell disorder affecting RBC, WBC, platelets	Paroxysmal cold hemoglobinuria
Chronic hemolysis of the disease associated with iron deficiency	Paroxysmal cold hemoglobinuria
Cold hemagglutinin associated with	IgM antibodies

Donath Landsteiner antibody (IgG)	Paroxysmal cold hemoglobinuria (cold agglutinin)

APLASTIC ANEMIA

Schwachmann diamond syndrome	**Bone marrow failure, pancreatic insufficiency, malabsorption**
Schwchmann diamond syndrome is associated with	*Exocrine pancreatic insufficiency*
Benzene is associated with	**Aplastic anemia**
Dyskeratosis congenita is associated with	**Aplastic anemia**
Chloramphenicol is associated with	Aplastic anemia
Aplastic crisis in chronic hemolytic anemia is precipitated by	Parvovirus B19
Parvo virus B19 infection is associated with	**Giant pronormoblast**
Acquired aplastic anemia caused by	Whole body irradiation, Primary stem cell defect, Chloramphenicol
Bone marrow aplasia is caused by	Chloramphenicol, alphamethyl hydantoin, phenylbutazone
Diseases causing aplastic anemia	PNH, hepatitis, pregnancy, chloramphenicol, HIV 1, parvovirus
Seen in COPD	Aplastic anemia
Acquired aplastic anemia NOT seen in	Fanconi's syndrome
NOT a cause of acquired pure red cell aplasia	Drug induced by NSAID
NOT a cause of aplastic anemia	Hepatitis A
25 year female, complaints of fever and weakness. Splenomegaly 3 cm below costal margin. Hb 8 mg/dl, TLC 3000/mm3,platelet count is 80,000/mm3	Aplastic anemia
Aplastic anemia can progress to	PNH, AML, myelodysplastic anemia
NOT seen in aplastic anemia	Splenomegaly
Treatment of Severe aplastic anemia	Anti thymocyte globin followed by cyclosporine
Most effective treatment of severe hypoplastic anemia in 20 year adult male	Bone marrow transplantation

PANCYTOPENIA AND FANCONI ANEMIA

Stem cells of bone marrow	Lymphoblast, Myeloblast, Normoblast
Hypersplenism is associated with	Pancytopenia
Pancytopenia with cellular marrow seen in	Megaloblastic anemia, myelodysplasia, PNH
Pancytopenia with cellular marrow is not seen in	G6PD deficiency
Congenital aplastic anemia	Fanconi anemia
Fanconi's anemia is caused by	Old degraded tetracycline
Pancytopenia with hypoplastic marrow	Fanconi's anemia
Fanconi's anemia	Hypocellular marrow, congenital anomalies, normocytic or macrocytic cell morphology
Fanconi anemia	Autosomal recessive, positive family history, inherited chromosomal instability syndrome
Patients with fanconi anemia are associated with risk of	AML and squamous cell carcinoma of skin, head and neck GIT, genital tract

Radial ray defects and hypoplastic thumb is associated with	*Fanconi anemia*
A male child presents with Fanconi anemia & hypercalciuria. Features of Dent's disease	Proteinuria, hypercalciuria, rickets

MYELODYSPLASTIC SYNDROME

MC cytogenetic change in Myelodysplastic Syndrome in adult	5q deletion
Cytogenetic change in Myelodysplastic Syndrome in children	Monosomy 7
Myelodysplastic syndrome	>50
Megaloblastoids are seen in	**Myelodysplasia**
Ringed sideroblast characteristically seen in	Myelodysplastic syndrome
Ringed sideroblast	**Mitochondria encrusted iron**
Pawn ball megakaryocytes is characteristic of	Myelodysplastic syndrome
International prognostic scoring system for	**Myelodysplasia**
NOT true about Myelodysplastic syndrome	Hypocellular bone marrow
Act by hypomethylation	Decitabine
Treatment of MDS with 5q deletion	*Lenalidomide*
Drug used for myelodysplastic syndrome	**Lenalidomide**

MYELOPROLIFERATIVE DISORDERS

Myeloproliferative disorder	Polycythemia vera, Myelofibrosis, Essenstial thrombocytosis, CML
NOT a myeloproliferative disorder	Mantle cell lymphoma
Transient Myeloproliferative disorder of Newborn is associated with	Down's Syndrome
NOT a laboratory diagnosis of differential diagnosis of chronic myeloproliferative disorder	Flow cytometry analysis

POLYCYTHEMIA

Viscosity of blood is increased in	Polycythemia, Multiple Myeloma
Causes of relative polycythemia	Dehydration, dengue hemorrhagic fever, Gaisbock syndrome
MC cause of secondary polycythemia in children	*R to L shunt*
Seen in infant of diabetic mother	Polycythemia
Criteria for diagnosis of Polycythemia vera	Elevated red cell mass, Increased counts of all cell lineages, splenomegaly
Major criteria for diagnosis of polycythemia vera	JAK 2 mutation
JAK 2 gene in	*Chromosome 9p*
32 year high altitude mountaineer. Hematocrit 70 %,	Polycythemia with increased red cell mass
Differentiate primary polycythemia from secondary polycythemia	Arterial oxygen tension < 90% on room air

Secondary polycythemia may be seen in	Cor pulmonale
Secondary polycythemia may lead to	Renal cell carcinoma, cerebellar hemangioblastoma, chronic cor pulmonale
Infantile Polycythemia	Cerebellar hemangioma
Polycythemia	Von Hippel Lindau Syndrome
Polycythemia vera	Decreased erythropoietin, Low ESR,Increased RBC mass, Increased Leucocyte alkaline phosphatase
Seen with polycythemia vera	Thrombosis, hyperuricemia, prone for acute leukemia
Tumour associated with polycythemia vera	Cerebellar hemangioma
High levels of LAP	Infection, Inflammatory disorder, growth factor therapy, pregnancy, OCP, stress, Myeloproliferative disorders EXCEPT CML, drugs
Highest level of leucocyte alkaline phosphatase	Polycythemia vera
Aquagenic pruritis	*Polycythemia vera*
Polycythemia vera	Increased leucocyte alkaline phosphate
Erythropoietin in polycythemia	Decreased
MC postoperative complication in Polycythemia vera	Uremia
MC complication after major surgery in polycythemia vera	Thrombosis
Elevated ESR is NOT seen in	Polycythemia rubra
Chuvash polycythemia is due to	**VHL mutation**
NOT commonly seen in polycythemia vera	Spontaneous severe infection
NOT seen in polycythemia vera	Thrombocytopenia
NOT a feature of Polycythemia vera	Increased erythropoitein
Does NOT occur in polycythemia	**Iron overload**
Most relevant investigation to rule out Polycythemia vera	Red cell mass
Dose of radioactive phosphorus to induce remission in Polycythemia	7 millicuries
Radioactive phosphorus is used in treatment of	Polycythemia
Radioisotopes are NOT used in treatment of	Secondary Polycythemia

MYELOFIBROSIS

Leucoerythroblastic picture	Myelofibrosis, Metastatic carcinoma, Gaucher's disease
A patient of myeloproliferative syndrome presents with decreased white cell count and decreased platelets	Myelofibrosis
Thrombocytosis is seen in	Myelofibrosis
Massive splenomegaly	Myelofibrosis
Grey platelet syndrome is due to defect in	*Alpha granules*
Grey platelet syndrome is associated with	**Myelofibrosis**
Dry marrow tap, tear drop cells	Myelofibrosis
Hb-6gm%,TLC 1200,platelets 60,000, MCV 12fL	Myelofibrosis
Confirmation of diagnosis of Myelofibrosis	Bone marrow biopsy

ESSENTIAL THROMBOCYTOSIS

Erythromelalgia due to	**Thrombocytosis**

Essential thrombocytosis also known as	Essential thrombocythemia
Symptom of Essential Thrombocytosis	Early Bruising
Splenomegaly least likely associated with	Primary thrombocytosis
Treatment of essential thrombocytosis	Anagrelide or Interferon alpha

WHITE BLOOD CELLS

GENERAL FEATURES OF WHITE BLOOD CELLS

Block at promyelocytic stage causes	**Severe congenital neutropenia**
Block at myelocytic stage causes	**Warts, hypogammaglobulinemia, immunodeficiency, myelokathexis**
True statement about white blood cells	Lymphocytes are important protective influence against childhood infections. After puberty, neutrophils replace lymphocytes as the predominant leucocyte
Round cell having fine nuclear chromatin, prominent nucleoli and fine azurophilic granule	Myeloblast
Arbiskow cells	*Myeloblastoma*
Leucocytosis is NOT seen in	Typhoid
Leucopenia may be a side effect of	Ara C (cytarabine)
Filgastrim	Recombinant G-CSF
G-CSF and GM-CSF in hematopoiesis cause	Leucocytosis
Anti GM-CSF antibodies associated with	Pulmonary alveolar proteinosis
GM-CSF cause	Capillary leak syndrome
Leukemoid reaction	*High LAP score, neutrophil > 11,600/ml, presence of mature neutrophil precursors in smear*
Leukemoid reaction is seen in	Acute infection
Leukoerythroblastic picture seen in	Myelofibrosis, metastatic carcinoma, Gaucher's disease
Leucoerythroblastic reaction is seen in	Secondaries in bone, Multiple myeloma, Lymphoma, myelofibrosis, Gaucher's disease
Leucoerythroblastic reaction NOT seen in	Lymphoma
Tumors that can be regressed by splenic irradiation	**CLL, PML**
Abscopal effect	**In treatment of metastatic cancer, localized irradiation of tumor not only causes regression of irradiated tumor but also regression of tumors far from irradiated site**
Abscopal therapy is associated with	*Spleen*

NEUTROPHILS

Biochemical features of neutrophil	*Active glycolysis, active pentose phosphate pathway, rich in lysosomes, contains CD11/CD18 integrins in plasma membrane*
Life span of neutrophils	6 hours

Mediators of neutrophils	Elastase, Cathepsin, Nitric oxide, Leukotrine
Found in secondary granules of neutrophil	Lactoferrin
Neutrophil secretes	Myeloperoxidase, Cathepsin G
Lactoferrin	Present in secondary granule of cytoplasm, present in exocrine secretion, great affinity for iron
Secondary granule seen in neutrophil	Lactoferrin
NOT a secondary granule of neutrophil	*Myeloperoxidase*
Dohle bodies	Neutrophils
Associated with drug induced neutrophilia	Steroids
Agranulocytosis means	Decrease in neutrophil count
Neutropenia is caused by	Cephalosporins, Ranitidine, Phenytoin
Reverse isolation is used for	*Neutropenia*
Filgastrim is used in treatment of	Neutropenia
Sustained neutropenia seen with	Carmustine
Treatment of Kostmann syndrome (severe congenital neutopenia)	G-CSF
Neutropenia is NOT caused by	Glucocorticoids
NOT essential for febrile neutropenic patient	White cell infusion

EOSINOPHILS

Eosinophil secrete	Major basic protein, Reactive form of O2, Eosinophilic chemotactic factor
Eosinophil does NOT secrete	Hydrolytic enzyme
Drug of choice for tropical pulmonary eosinophilia	DEC
Eosinophilia is associated with	Coccidiomycosis
Causes of eosinophilia	Hodgkin's disease, filariasis, HIV
Condition NOT associated with Eosinophilia	Amoebiasis
Hypereosinophilic syndrome is characterized by	*Presence of more than 1500 eosinophils per microliter of peripheral blood for 6 months or longer*

MONOCYTE

Half life of Monocytes in circulation	1-3 days
Monocytes are produced by	Bone marrow & Lymph node
Absolute monocytosis is seen in	Kala azar, TB, Brucellosis

LYMPHOCYTES

Flow cytometry is done for	Lymphocyte
Absolute lymphocytosis	Infectious mononucleosis, Whooping cough
Autoimmune lymphoproliferative syndrome	*Canale smith syndrome, chronic non malignant lymphoproliferation, disorder of abnormal lymphocyte apoptosis*

LEUKOCYTE ADHESION DEFICIENCY

Leucocyte adhesion deficiency is associated with	**Mutation in CD 18**
Most common cause of umbilicus does NOT separate at age of 2 years	Leucocyte adhesion deficiency
Leucocyte adhesion deficiency	Defective Neutrophil fusion
Lack of leucocyte adhesion molecule is associated with	Delayed closure of umbilical cord
Leucocyte adhesion deficiency is associated with	*Gingivitis, neutrophilic enzyme deficiency*
Defect in Leucocyte Adhesion Deficiency Type I	α-Interferon
LAD 1 is due to deficiency of	*CD 11a : CD 18*
LAD 1	**Delayed separation of umbilical cord**
LAD 2 (also known as congenital disorder of glycosylation IIC)	**Mental retardation**
Leucocyte adhesion deficiency type II due to defect in	*Fucosyl transferase*
LAD 3	**Petechial hemorrhage**
Leucocyte adhesion deficiency type III is associated with	**Kindlin**

GENERAL FEATURES OF LEUKEMIA

MC type of congenital leukemia	*AML*
Acute Leukemia may be caused by exposure to	Benzol
Benzene associated with	Leukemia
Secondary leukemia associated with	Cisplatin
Conditions predisposing to Leukemia	Myelofibrosis, Ionising radiation, Mycythemia Vera
Genetic risk factors for leukemia	Down's syndrome, Bloom's syndrome, Ataxia telangiectasia
Condition NOT predisposing to Leukemia	Infectious mononucleosis
NOT a preleukemic condition	Paroxysmal cold hemoglobinuria
LEAST likely pre leukemic condition	Paroxysmal cold hemoglobinuria
Acute leukemia, CD19+ve, CD10+ve, CD33+ve, CD13+ve	Biphenotypic leukemia
CD19 +ve,CD117+ve,MPO+ve,CD3-ve.CD33-ve	Mixed phenotypic leukemia
A child with TLC-60000, Hb-9.1, CD10+, cd33-, MPO+, CD117+, CD19-	Multi lineage leukemia
Chronic neutrophilic leukemia is associated with	**T(15,19)**
Classification of B cell lymphoid leukemia	**RAI system, Binet system**
5 year old child, fever on and off, petechial spots all over the body, increasing pallor for past 1 month. Splenomegaly of 2 cm below costal margin	Acute leukemia
Earliest feature of childhood leukemia	Radioluscent transverse metaphyseal band
Most characteristic radiographic sign in a child with leukemia	Metaphyseal transuluscencies
X ray finding in acute leukemia	Osteolytic lesion in flat bones, Periosteal new bone formation, Osteosclerosis of long bone, Transverse line of dark band below the growth plate
X ray features of leukemia in a 2 year old child	Sub periosteal erosions, Metaphyseal Osteoporosis, Thick line below growth plate

Drug of choice in eosinophilic leukemia	Corticosteroids
Used for leukemia causing chest pain, pulmonary infiltrates and pleural effusion	Tretinoin
Complication of treatment of leukemia	**Cell lysis pneumopathy**
Main pathology in leukemic cell lysis pneumopathy	*Pulmonary hemorrhage*

ACUTE LYMPHOBLASTIC LEUKEMIA

MC Malignancy in Children	ALL 1st, 2nd Brain tumour, 3rd Neuroblastoma, 3rd MC in Children > 1 year Wilm's Tumour
MC leukemia in children	ALL
MC type of ALL	Pre B cell ALL
MC type of ALL & best Prognosis	L1
Rarest type of ALL & Worst Prognosis	L3
Testicular involvement in ALL	T type
Precursor T cell ALL	Immature T cell phenotype Tdt/CD34/CD7
Mediastinal Widening	T cell ALL
True about ALL	TdT positive
Presents as mediastinal involvement	ALL
Down's Syndrome more prone to develop	ALL
Mediastinal widening is seen in	T cell ALL
Mediastinal enlargement is caused by	Lymphoblastic leukemia
Congenital chromosomal abnormality is seen in	ALL
Auer rods	ALL
Auer rod represent	Fusion of Primary Lysosomal granules
Blast cells of ALL in childhood contain	CALLA antigen
ALL L3 arise from	Mature B cell
Acute lymphoblastic leukemia with hypereosinophilic syndrome	Eosinophils are NOT a part of neoplastic clone, t(5,14) rearrangement may be detected in blasts, peripheral blood eosinophilia may normalize with chemotherapy
Negative for MPO and Non specific esterase. positive for CD 19,CD 10,CD 22,CD 20	Acute lymphoblastic leukemia with hypereosinophilic syndrome
Mediastinal lymphadenopathy in a young boy	T cell lymphoblastic ALL
T(2,8) associated with	B cell ALL
T(17,19)	ALL
T(9,22)	ALL
CD10 is seen in	ALL
Acute lymphoblastic leukemia of T cell lineage	An immature T cell phenotype TdT,D34,CD7 positive
Poor prognostic factor in ALL	Hypoploidy
Poor prognosis in ALL	Intrathoracic mass, Cranial secondaries
Poor prognostic factor in ALL	T (9,22) t(4,11)
Poor prognostic factor in ALL	Cranial secondaries, Age 2 to 10 years, Intrathoracic mass, Trisomy 8
Poor prognostic factor for ALL	Hypodiploidy
Poor prognostic factor for ALL in children	Testicular involvement
Worst Prognosis in Childhood Leukemia	Precursor B ALL
NOT having good prognosis	Pre B cell tumour
NOT a good prognostic factor of ALL	T 9,22 8,14 4,11

Good prognostic factor of childhood ALL	Hyperploidy, Female sex, t(12,21) translocation
Good prognostic factors of ALL	*Age of onset 2 – 8 years, WBC intake < 50,000*
PAS stain shows black positivity in	Lymphoblast
Vincristine	Alkaloid, alopecia, neurotoxicity, useful for induction of remission in acute lymphoblastic leukemia
Treatment of choice in intracranial ALL	Intrathecal methotrexate
Acute leukemia patient admitted with febrile neutropenia, on day four of being treated with broadspectrum antibiotics his fever increase. X ray show bilateral fluffy infiltrates. next step	Add cotrimoxazole
L asparaginase is particularly used in	ALL
Prophylactic methotrexate is administered for	ALL
Lymphocytopenia seen a few hours after administration of a large dose of prednisolone to a patient with lymphocytic leukemia is due to	Massive lymphocytic apoptosis
Drug NOT used in management of ALL	All trans retinoic acid

ACUTE MYELOBLASTIC LEUKEMIA

MC type of AML	M2
MC type of AML in Down's Syndrome	M7
Auer rods are typically NOT seen in	M0
Presents as chloroma	*M2*
DIC seen in	M3 (acute promyelocytic leukemia)
Auer rods are frequently seen in	M3
pathological mechanism leading to acute promyelocytic leukemia	*Abnormal gene fusion*
AML with gum infiltration and splenomegaly	M4 (myelomonocytic)
M4	*Peroxidase positive, NSE positive, inversion 16*
M5	*Peroxidase negative, NSE positive, organomegaly, gum infiltration*
M5	**Monocytic**
Focal gingival swelling is associated with	**Acute myelomonocytic leukemia**
All trans retinoic acid used in treatment of	Acute promyelocytic leukemia
PML	Retinoic acid is used in treatment, associated with DIVC,15/17 translocation
Sheet of atypical plasma cells`	Promyelocytic leukemia`
Arsenic used in treatment of	Acute promyelocytic leukemia
Non specific esterase is NOT positive in	M0,M1,M2,M6
Non specific esterase in AML is negative in	M6
M6	**Erythroleukemia (Di gugliemo disease)**
M7	**Megakaryocytic**
Characteristically lead to marrow fibrosis	*Acute megakaryocytic leukemia*
Down's syndrome is associated with	*AML 7*
Prognosis of AML in Down's syndrome is better than	*That of normal individuals*

AML is characterized by	Auer rods in blast cells
Blueberry muffin is associated with	*AML*
Syndromes associated with AML	Down's syndrome, Klinefelter syndrome, Patau syndrome
Blood picture with t(8:14), low white cell and platelets, 10 % blasts	AML
Bone marrow aspirate was dry, bone marrow biopsy was possible-acute leukemia with myelofibrosis	Megakaryocytic AML
Faggot cells	Cells with Multiple Auer Rods, M3 Type of AML
T(8,21)	AML with Maturation M2
T(15,17)	AML M3
T(9,11)	AML Acute Monocytic M5
T(15,17)	AML Acute Promyelocytic M3
Inversion 16 associated with	AML
Granulocytic Sarcoma (Chloroma, AML away from Marrow)	117
Poor prognostic factors	Age more than 60, leucocytes > 1 lakh/microlitre, secondary leukemia, Complex karyotype, AML M7, deletion 7q
AML with worst prognosis	Monosomy 7
Prognosis in AML indicated by	Monosomy 7
Auer rods	Poor prognosis
Poor prognostic feature	*Deletion of chromosome 5q*
NOT a poor prognostic feature of AML	Presence of t(8,21)
Inversion 16	Good prognosis
MDRI gene	Good prognosis
Favorable mutation in AML	**NPM1, CEBPA**
2 week history of weakness and bleeding gums, pancytopenia. 26% blasts. Auer rods mature myeloid cells. Occasional neutrophil with pseudo pelger huet anomaly	Myeloperoxide positive
Acid phosphatase is specific to	Monocyte
Treatment of AML child with hyperleucocytosis	IV fluids, allopurinol, alkalinisation
In case of CNS relapse in AML, chemotherapy would consist of	Intrathecal methotrexate + cytosine arabinoside
Arsenic useful in treatment of	Acute promyelocytic leukemia
Acute promyelocytic leukemia differentiation syndrome	*Occurs within 3 weeks of treatment, known as retinoic acid syndrome, related to adhesion of differentiated neoplastic cells to pulmonary vasculature endothelium, discontinuation of tretinoin is required in severe cases*

CHRONIC LYMPHOCYTIC LEUKEMIA

Average age of onset of CLL	50 – 60
CLL is usually of	B cell variety, Elderly age
Translocation associated with CLL	**(14,15)**
Autoimmune hemolytic anemia is commonly seen in	CLL

Leukemia associated with autoimmune hemolytic red cell aplasia	*CLL*
80 year old symptomatic man, TLC one lakh, 80% lymphocytes, 20% PMC	CLL
Only Leukemia NOT associated with Ionizing radiation	CLL
CLL	Can present as acute leukemia, leukocytosis is prominent, diagnosed on routine blood tests
CLL	Trisomy 12 correlates with aggressive clinical course, abnormalities of 13q14 associated with long term survival,11q22-23 deletions have excessive lymphadenopathy
Diffuse effacement by a predominant population of small lymphocytes	CLL
Proliferation centres/pseudofollicular growth centres	CLL/SLL
MicroRNA is involved in	*CLL*
CLL	Treatment is not needed in asymptomatic patients
NOT true about CLL	T lymphocyte CLL is common
CLL characteristically positive for	CD 5,19,23
CLL	5,23 positive
CLL	CD 5,19,22 positive
CLL negative for	CD 79B,CD22 FMC7
Alemtuzumab used in CLL targets	*CD52*
Cyclin dependent kinase inhibitor under clinical development for treatment of CLL	*Flavopiridol*

CHRONIC MYELOCYTIC LEUKEMIA

Classification of CML	**Solkal index, Hassford system**
Chronic myelomonocytic leukemia	Peripheral blood monocytosis > 10^9/L, Absence of Philadelphia chromosome, Less than 20% of blasts in blood or bone marrow, Absent or minimal dysplasia of myeloid lineage
Vitamin B12 level in chronic myelomonocytic leukemia	*Increased*
Leukemia cutis is seen in	*Chronic monocytic leukemia*
Peripheral smear with increased neutrophils, basophils and eosinophil, platelets are highly suggestive of	Chronic myelogenous leukemia
In a patient with CML, hb falls from 11g% to 4 g% in a short span of time and splenomegaly occurs	Accelerated phase, Blast crisis
Vitamin B12 level in CML	Increased
Basophilic leucocytosis occur in	CML
CML in children is associated with	Down’s syndrome
CML is characterized by	Decreased Leucocyte Alkaline Phosphate
Platelets are always elevated in	*CML*
Prognosis in CML	Percentage of blasts, spleen size
Philadelphia chromosome is seen in	CML
ABL (Translocation)	CML, ALL
T(9,22)	CML, ALL
First reproduced chromosomal anomaly	**T(9,22)**

ABL on	**Chromosome 9**
NOT a feature of juvenile CML	Philadelphia chromosome is positive
NOT compatible with diagnosis of CML	More than 20% blasts in blood or bone marrow
NOT compatible with diagnosis of juvenile myelomonocytic leukemia	Presence of bcr/abl fusion gene
NOT a criteria for diagnosis of chronic myeloid leukemia in accelerated phase	Persistent thrombocytosis unresponsive to therapy
Drug of choice for CML	Busulfan
Side effect of Busulfan	*Addisonian like picture*
Drug of choice for CML	Imatinib mesylate
Drugs approved for CML	**Dasatinib, Nilotinib**
If patients are intolerant to tyrosine kinase inhibitors	**Omacetaxine (derived from cephalotaxus harringotonia)**
Treatment of Choice for CML	Allogenic Stem Cell Transplantation
Drug decreasing tumor cells in autologous stem cell transplantation	**Hydroperoxycyclophosphide**
Common source of skin infection after hematopoietic stem cell transplantation	**Less than 1 month – HSV, 1 – 4 months – HHV – 6, >6 months – VZV**
Drug of choice for bacterial prophylaxis for hematopoietic stem cell transplantation	*Levofloxacin*

GENERAL FEATURES OF LYMPHOMA

Smoking does NOT predispose to	Lymphoma
25 year old man. CT show bilateral upper lobe fibrosis. Mediastinal enlarged lymph node with peripheral rim enhancement	Lymphoma
MC type of lymphoma presenting as mediastinal mass	*T cell lymphoma*
MALTOMA is	B cell lymphoma
Lymphoma associated with HTLV infection	Adult T cell leukemia and lymphoma
Plasmocytoid lymphoma may be associated with	IgM
Waldenstrom's Macroglobulinemia is seen in	Plasmocytic Lymphoma
In Waldenstrom's macroglobuinemia, increased levels of	IgM
Milder form of waldenstorm macroglobulinemia	**Cold agglutinin**
Having a shorter survival	Primary effusion lymphoma
Involvement of other organs by GI lymphoma	Stage IV
Mass at duodenojejunal flexor involving renal papillae. HPE lymphoma	IV E stage
NOT a B cell neoplasm	Angiocentric lymphoma
International prognostic index for lymphoma	Stage of disease, number of extralymphatic site involved, LDH, age
NOT a prognostic factor in 'International Prognostic Index" for lymphoma	Hemoglobin and albumin
Hypercalcemia in lymphoma is due to	*1,25 dihydroxy vitamin D*
Pt >60 yr leukocyte= 20,000 with generalized lymphadenopathy Next test you should do?	Peripheral blood immunohistochemistry
B symptoms are common in	**Anaplastic large T cell lymphoma**
Anaplastic large T cell lymphoma	**CD 30 +, (2,5) translocation**

Latest treatment for anaplastic large T cell lymphoma	*Crizotinib (ALK inhibitor)*
Villous lymphocytes are associated with	*Splenic marginal zone lymphoma*
MALTOMA skin is associated with	*Borrelia*
MALTOMA eyes associated with	*Chlamydophilus psittaci*
MALTOMA small intestine is associated with	*Campylobacter*
Lymphoma like diseases	**Rosai dorfman syndrome (sinus histiocytosis with massive lymphadenopathy), lymphomatoid papulosis, Castleman disease**
Characteristic feature of Rosai Dorfman disease	*Emperopolesis*

HODGKIN’S LYMPHOMA

MC type of Hodgkin’s Lymphoma	Nodular Sclerosis
MC type of Hodgkin’s Lymphoma in India	Mixed Cellularity
Popcorn Cells	Lymphocyte Predominant Hodgkin’s Lymphoma
Popcorn variant of Hodgkin’s lymphoma	*CD 15 negative, CD 20 positive, CD 45 positive*
Hilar lymphadenopathy is seen in following hodgkin’s lymphoma	Nodular sclerosis
Hodgkin’s lymphoma with best prognosis	Lymphocyte predominance
Subtype of Hodgkin’s lymphoma which is histogenetically distinct from other subtypes`	Lymphocyte predominant
Lymphocytic and histiocytic variant of Reed Sternberg cell is seen in	Lymphocyte predominant Hodgkin’s disease
Excellent Prognosis Hodgkin’s Lymphoma	Lymphocyte predominance >Nodular Sclerosis
Hodgkin Lymphoma showing biphasic incidence	Mixed cellularity
Lowest number of RS cells	Lymphocyte rich
Hodgkin’s lymphoma with Best Prognosis	Lymphocyte Predominance
Hodgkin’s Lymphoma with Worst Prognosis	Lymphocyte Depletion
Non Classical Hodgkin’s Lymphoma	Lymphocyte Predominance
Lacunar cells	Hodgkin’s lymphoma of sclerosis nodular subtype
Progressive transformation of germinal centres (PTGC) is a precursor lesion for	Hodgkin’s lymphoma Nodular sclerosis
45 year lady presents with painless supraclavicular lymphadenopathy, biopsy revealed binucleated acidophilic owl eye appearance with floating lymphocyte in empty space with CD 15, CD 30 positive. Probable diagnosis	Nodular sclerosing lymphoma
L and H cells in seen in	Lymphocyte predominant variant
Reed Sternberg cells	Hodgkin’s Lymphoma
Reed Sternberg Cells (Classical Hodgkin’s Lymphoma)	15,30
Reed Sternberg cells are characteristic of	Hodgkin’s lymphoma
Reed Sternberg cell is derived from	B cell
Hodgkin’s lymphoma most commonly involves	Neck nodes
MC Site of Lymph Node Enlargement in Hodgkin’s Lymphoma	Mediastinal

MC Presentation of Hodgkin's Lymphoma	Painless Enlargement of Lymphnode
Most common site of enlargement of lymph nodes in Hodgkin's lymphoma	Mediastinal
Fever for 3 – 10 days and afebrile for 3- 10 days	**Pel ebstein fever (Hodgkin's lymphoma)**
Paraneoplastic syndrome associated with Hodgkin's disease	Cerebellar degenerative disease
Persistent fever, significant weight loss for past two months. On examination he is observed to have cervical and axillary lymphadenopathy. Investigations are remarkable. Biopsy from cervical nodes is performed and returns positive for Hodgkin's lymphoma.	Stage II B
NOT true about Hodgkin's lymphoma	CNS is the most common site
NOT true about Hodgkin's Lymphoma	Osteolytic metastasis
Epithelioid granuloma is NOT seen in	Hodgkin's lymphoma
NOT a common lymphoma of orbit	Hodgkin's lymphoma
Good prognostic feature of Hodgkin's disease	Hemoglobin >10 gm/dl, WBC count < 15000/mm3, age < 45 years
Poor prognostic factors of Hodgkin's lymphoma	Systemic manifestation, lymphocyte depletion, mediastinal disease, stomach involvement Absolute lymphocyte count <600 /microliter
Diagnosis of hodgkin's disease is confirmed by	CT scan
CD30 associated with	Lymphoma
Bone marrow study is best done in	Hodgkin's Lymphoma
Diagnosis of Hodgkin's lymphoma is NOT based on	Atypical cells in background
Total dose of radiation in Hodgkin's disease	3000 – 5000 rad
Treatment of choice for stage III a Hodgkin's lymphoma	Combination of chemotherapy and radiotherapy
Drugs used for Hodgkin's Lymphoma	Adriamycin, Bleomycin, Vinblastine, Dacarbazine
Treatment of nodular sclerosis type of hodgkin's lymphoma with lacunar cells with isolated cervical lymphadenopathy	Radiotherapy alone
Treatment of Hodgkin's IA	Radiotherapy is best
Risk of infertility in Hodgkin's lymphoma is more with	MOPP regimen
Hodgkin's lymphoma	ABVD is more commonly used regimen, combination chemotherapy is main stay of treatment in advanced Hodgkin's disease, WBC count > 15000/mm3 is a poor prognostic factor
Stanford V regimen for	**Hodgkin's lymphoma**
Mantle irradiation is used in	*Hodgkin's lymphoma*

NON HODGKIN'S LYMPHOMA

Classification for Non Hodgkin's lymphoma	**Rappaport classification, Luke classification**
Classification proposed by international lymphoma study group for Non Hodgkin's lymphoma	REAL classification
MC type of NHL in children	*Burkitt's lymphoma*
MC type of NHL in Indian children	*Lymphoblastic leukemia*
Staging of NHL in children	*Saint Judge staging*
MC type of NHL worldwide	Diffuse large B cell lymphoma
Diffuse large cell lymphoma has worse	*Follicular lymphoma*

prognosis than	
Diffuse large cell lymphoma	**Bcl 2, Bcl 6**
Type of diffuse large B cell lymphoma with good prognosis	*Germinal centre B cell like*
Intermediate form of Non Hodgkin's lymphoma	Diffuse small cleaved cell
Most malignant form of Non Hodgkin's lymphoma	Diffuse large B cell
Low grade Non Hodgkin lymphoma	Follicular
Typical violaceous plum colored skin nodule	**Non Hodgkin's lymphoma of skin**
Non Hodgkin's lymphoma associated with hepatitis C virus	**Lymphoplasmocytic lymphoma (9,14)**
NOT true about Non Hodgkin's lymphoma	Follicular / nodular NHL has worse prognosis than diffuse NHL
Rituximab used in	NHL, RA, SLE

BURKITT'S LYMPHOMA

Highly malignant, rapidly progressive and delay in treatment of even 1-2 days can lead to death	*Burkitt's lymphoma*
Commonest tumor among children 1-5 years in south Africa	Burkitt's lymphoma
Virus associated with Burkitt's lymphoma	EBV
Pathognomic feature of Burkitt's lymphoma	Starry sky pattern
Feature of Burkitt's lymphoma	*Multiple small nucleoli, proliferation of macrophages*
NOT true about Burkitt's lymphoma	Radiotherapy is the treatment of choice
NOT a feature of Burkitt's syndrome	Polyclonal
c-MYC (Translocation) on chromosome 8	Burkitt's Lymphoma
T(2,8)(p12:q24)	Burkitt's Lymphoma
Bcl-6 associated with	Burkitt's lymphoma
Burkitt's Lymphoma	T(8,14)
Burkitt's lymphoma	CD 34 negative, surface immunoglobulin positive
Bcl 6	Burkitts lymphoma

HAIRY CELL LEUKEMIA

HTLV II has been implicated in causation of	Hairy cell leukemia
Hairy cell leukemia is a neoplastic proliferation of	B cells
Hairy cell leukemia	Splenomegaly is conspicuous. cells are positive for tartrate resistant acid phosphatase,CD25 expression
Hairy cell leukemia presents with	Splenomegaly
Massive splenomegaly	Hairy cell leukemia
Characteristic expression of Hairy cell leukemia	CD 11c,CD25,CD103
Hairy Cell Leukemia	CD 19,22,103
Bone marrow biopsy absolutely indicated in	Hairy cell leukemia
Drug of choice in hairy cell leukemia	Cladrabine
Drug used in hairy cell leukemia	Cladrabine, pentostatin, interferon alpha
NOT a treatment of Hairy cell leukemia	Splenectomy
NOT used in treatment of hairy cell leukemia	Steroid

MANTLE CELL LYMPHOMA

63 year male, massive splenomegaly, lymphadenopathy, TLC 1,70,000 per mm3,CD19 positive,CD5 positive,Cd23 negative, monoclonal B cells with bright kappa positively comprising 80% of peripheral blood lymphoid cells	Mantle cell lymphoma
CD 10 associated with	Mantle cell lymphoma
T(11,14)	Mantle Cell Lymphoma
Mantle cell Lymphoma	19,79B,FMC7,Bcl-1
Mantle cell lymphoma	CD 5,20,43

FOLLICULAR LYMPHOMA

Bcl-2 and t(14,18) associated with	Follicular lymphoma
T(14,18) is associated with	Follicular lymphoma

POST TRANSPLANT LYMPHOMA

Post transplant lymphoma occurs due to proliferation of	B cells
Post transport lymphoma	B cell
MC organism causing post transplant lymphoma	EBV

BLEEDING AND COAGULATION DISORDERS

GENERAL FEATURES OF BLEEDING AND COAGULATION DISORDERS

Hyperviscosity is seen in	Multiple myeloma, Cryoglobulinemia, Lymphoma, Macroglobulinemia
Increased viscosity & decrease in rate of circulation leads to	Rouleaux formation
Conditions associated with incoagulable state	Abruptio placenta, Acute promyelocytic leukemia, Severe falciparum malaria, Snake envenomation, Heparin overdose
MC cause of death in coagulation disorders	Cerebrovascular bleeding
25 year asymptomatic female underwent preop coagulation test. her BT is 3 minutes, PT 15/14 sec, aPTT is 45/35 sec. platelet count is 2.5 lakh, factor VII 60 IU/L. diagnosis	Factor VIII inhibitors
22 year female with recurrent joint pain developed petechial hemorrhage. most likely to have	Acquired factor VIII inhibitors
Patient has ecchymoses and petechiae all over the body with no hepatosplenomegaly.	Increased megakaryocytes in bone marrow, Decreased platelet count in blood, Disease resolves itself in 80% of patients in 2-6 weeks

PT 26, control PT 13 seconds, sensitivity index 1.41.INR	(26/13) 1.41
Increased INR on patient on warfarin is associated with	**Statin**
Bethesda assay is for	*Unexplained prolongation of PT, apTT*
Bethesday assay	*Clotting factor inhibitor assay*

GENERAL FEATURES OF PLATELETS AND ENDOTHELIAL CELLS

Endothelial cells secrete	vWF
Endothelial cells secrete	Thrombomodulin, heparin like molecules, secrete plasminogen activators, degrade ADP, synthesize protein S, PGI2, nitric oxide
Ultrastructurally endothelial cells contain	Weibel palade bodies
Endothelium derived growth factor is associated with	nNOS
vWF is synthesized in	Endothelial cells and megakaryocytes
Epitheloid hemangioendothelioma	*Expresses factor VIII antigen*
Alpha granules of platelet contains	vWF
Dense granules of platelet contains	ADP, Serotonin, Calcium
Storage of platelets	5 days,19-24 degrees
Platelet adhesion to collagen via	vWF
Thromboxane A2 is secreted by	Platelets
Thromboxane A2	Low dose aspirin inhibits its synthesis, causes vasoconstriction in blood vessels, causes bronchoconstriction
Line of Zahn	Platelet thrombi
Inherited platelet function disorders	Bernard Soulier syndrome, Glanzman thrombaesthenia, von willebrand disease, Wiskott aldrich syndrome
Raised platelet count	Post operatively, acute hemorrhage, splenectomised patient
Serious bleeding if platelet count is below	40000
Least associated with prolonged bleeding time	Coagulation factor deficiency
Platelet function assessed by	BT and platelet adhesion assay
Platelet function defects	Normal platelet count, prolonged bleeding time
Bleeding time assess	Function of platelets
PFA 100 for	*Platelet function abnormalities*
Platelet storage pool disorder	Autosomal dominant, abnormalities of platelet granule formation
Treatment of inherited platelet dysfunction	**Despropressin**

PATHWAYS OF COAGULATION

Blood within the vessels do NOT clot normally because	Vascular endothelium is smooth and coated with glycocalyx
Blood in the vessels normally does not clot because	Thrombin has a positive feedback on plasminogen
Anticoagulant preventing clotting of blood in normal vascular system	Antithrombin II
During hemostasis, platelet affects coagulation area	Platelet adhesion to exposed endothelium, Clot retraction, Activation of prothrombinase complex, Vasoconstriction

Blood coagulation	Factor X is a part of both intrinsic and extrinsic pathway, intrinsic pathway can be activated invitro, calcium is required in several steps of coagulation
NOT involved in local hemostasis	Vitamin K
Prothrombotic	Adenosine Bisphophate
Activation of Extrinsic pathway	Tissue thromboplastin
Isolated prolongation of prothrombin time	Factor VII deficiency
Must for prothrombin time	Thromboplastin
Estimation of PT is NOT useful of	IX
Prothrombin time should be done within	2 hours
Prothrombin time depends on	2,5,7,10
Extrinsic pathway	Factor 7
Prothrombin time detects a deficiency of	VII
NOT involved in intrinsic pathway	VII
Elevation in both PT and APTT	Defect in common pathway
Prolongation of both PT and APTT	Deficiency of factor II,V,X
Partial thromboplastin time correlate with	Intrinsic and common pathway
Blood coagulation profile with prolonged partial thromboplastin time who can be allowed to undergo an operation without any intervention?	Factor XII deficiency
APTT is prolonged in deficiency of	8,9,11,12
Intrinsic pathway	8,9,11,12

CLOTTING FACTORS

Vitamin K dependent Clotting Factors	2,7,9,10
Vitamin K dependent clotting factors with shortest t ½	*Factor 7*
ERGIC 53 mutation	**Combined factor 5 and 7 deficiency**
Prothrombin is synthesized in	Liver
Procoagulation protein	Thrombin
Conversion of fibrinogen to fibrin is stabilized by	Thrombin
Thrombin activity is inhibited by	Alpha 2 macroglobulin >> heparin cofactor II
Increased aPTT and PT with no bleeding tendency	V
Labile factor is	*Factor V*
Conversion of prothrombin to thrombin require	Factor V and Ca++
Parahemophilia is associated with	*Factor V deficiency*
Substance present in both serum and plasma	Factor 7
VII	Proconvertin, Stable Factor
Factor VII	Deficiency is inherited as autosomal recessive trait, managed by fresh frozen plasma, shorter half life compared to Hageman factor XII
Tissue thromboplastin activate	Factor VII
Factor 8 is produced by	endothelium
Clotting factor NOT completely synthesized in liver	Factor VIII
Half life of factor 8	8 hours
An unit of whole blood that has been stored for 24 hours will have normal level of factors except	*Factor VIII*

Converging point of both pathway in coagulation	Factor X
Rosenthal syndrome is seen in deficiency of	XI
Invitro coagulation is initiated by	Factor 12
Factor XII	**Hageman factor**
Prolonged partial thromboplastin but normal prothrombin time	Factor XII deficiency
Urea clot lysis test positive with	Factor XIII deficiency
A newborn presented with profuse bleeding from umbilical stump after birth. probable diagnosis	Factor XIII deficiency
Cross linking and stabilization of clot	XIII
Bleeding from umbilical stump, urea clot lysis test positive	Factor XIII
Which helps in bridging fibrin in a clot and stabilizes the clot	Factor 13

THROMBOMODULIN

Binds with Prothrombin to cause anticoagulation by activating Protein C	Thrombomodulin
Thrombomodulin thrombin complex prevents clotting because	The complex removes thrombin and also activates protein C which inactivates the activated factors V and VIII
Thrombomodulin I is produced by	Splanchnic circulation, Skin circulation, Muscle circulation
Thrombomodulin I is NOT produced by	Cerebral circulation
Endothelial cells NOT producing thrombomodulin is seen in	Cerebral microcirculation

HEMOPHILIA

Inheritance of Hemophilia A & B	X linked recessive inheritance
Hemophilia affected man marries a woman	No males are affected, all females are carriers
Woman's brother and son having hemophilia	50% of sons affected
26 year female, PPH on second postnatal day, APTT and PTT are prolonged. BT, PT, platelet count are normal	Acquired hemophilia
Clotting time in Hemophilia	Prolonged
Feature used to differentiate hemophilia from von willebrand disease	Bleeding time
MC site of pseudotumour like growth in hemophilitic arthropathy	Iliopsoas
Hemophiliac pseudotumor is common in	Ilium
Hemophilic knee joint	Subchondral cyst formation, Widening of intercondylar notch, Squaring of patella
MC site of pseudotumor like growth in hemophilic arthropathy	Quadriceps
Spontaneous muscle bleeding typically seen in	Hemophilia
Accelerated Growth & Maturation of Epiphysis & Widening of Epiphysis	Hemophilia
DIC does NOT occur in	Hemophilia

Swelling of one knee joint. NOT a differential diagnosis	Hemophilia
NOT true about dental extraction in hemophilia patient	Extraction should be done under general anaesthesia and skilled anesthetic care
NOT true about hemophilia	Increased bleeding time
NOT a feature of hemophilic knee joint	Juxta articular osteosclerosis
Arthroscopy is contraindicated in	Hemophilia
Prenatal diagnosis of hemophilia by	*Genetic linkage analysis*
Prenatal diagnosis of hemophilia is best done by	PCR
Treatment of Hemophilia	Cryoprecipitate
Aminocaproic acid recommended for a hemophilic child with	Oral bleeding
Treatment of choice in hemophiliac with spontaneous hematuria	Prednisolone
Used in mild hemophilia	DDAVP
Treatment of hemarthrosis	Aspiration, POP , Traction, Compression bandage
MC mutation in hemophilia A	*Inversion of intron 22*
Hemophilia A	PTT increased, clotting time increased, serum levels of factor VIII decreased
Hemophilia is due to deficiency of	Factor 8
Classical hemophila due to deficiency of	8
Activity of factor VIII procoagulant is deficient in	Hemophilia
Cause of bleeding in hemophilia A	Lack of reaction accelerator during activation of factor X in coagulation cascade
Hemophilia A	Factor 8 deficiency
NOT true about Hemophilia A	PT increased
NOT a diagnostic feature of hemophilia A	Increased prothrombin time
Hemophilia B	Factor 9 deficiency

VON WILLEBRAND DISEASE

Major function of Von willebrand factor	Platelet adhesion
MC cause of acquired vWD	**MGUS**
Von willebrand disease	Autosomal dominant, Increased bleeding time, Factor VIIIc are decreased in circulation, APTT is increased
MC form of vWF disease	*Type 2A*
Most severe form of vWF disease	*Type 3*
Milder form of vWF disease	*Type 1*
Qualitative defect of vWF	vWD 2
Inheritance of vWD type I,II	Autosomal dominant
Bleeding time prolonged in	vWD
Von willebrand disease associated with	Normal prothrombin time
Coagulation profile in a 13 year old girl with menorrhagia having Von willebrand disease	Isolated prolonged PTT with normal PT
7 year old girl, repeated episodes of bleeding into joints. APTT is prolonged, PT is normal.	Von willebrand disease
8 year old with swelling of right knee joint, NOT a differential diagnosis	Von Willebrand disease
NOT true regarding von willebrand disease	Platelet count is abnormal
NOT true about von willebrand factor	Synthesized by hepatocytes

NOT true about von Willebrand disease	Increased platelet aggregation in response to riscocetin
Best assay for deficiency of von Willebrand disease	BT + APTT+ VWF - riscocetin factor assay
Test abnormal in both classical von willebrand disease and hemophilia A	*PTT*

GLANZMANN THROMBAESTHENIA

Disorder of Platelet aggregation	Glanzmann's thrombaesthenia
Riscocetin in Glanzmann thrombaesthenia	*Normal*
GpIIB/IIIA inhibitor	Abciximab, Eptifibatide, Tirofiban

BERNARD SOULIER SYNDROME

Bernard soulier disease is also known as	*Giant Soulier syndrome*
Gp Ib deficiency	Bernard Soulier Syndrome
NOT true about Bernard Soulier syndrome	Ristocetin aggregation normal

WISKOTT ALDRICH SYNDROME

Inheritance of Wiskott Aldrich syndrome	X linked recessive
Wiskott Aldrich Syndrome	Thrombocytopenia, Eczema, Recurrent Infections
Wiskott Aldrich syndrome	Bloody diarrhoea during infancy, Low IgM and elevated IgA and IgE (paradox), Atopic dermatitis
Presence of small sized platelets in peripheral smear	Wiskott Aldrich syndrome
NOT true about Wiskott aldrich syndrome	Autosomal recessive

THROMBOCYTOPENIA AND PURPURA

Thrombocytopenia is platelet count less than	One lakh
Bone likely to be absent in inherited thrombocytopenia	*Radius*
MC Coagulopathy in Surgical Patients	Thrombocytopenia
Kasabach Meritt syndrome	Platelet sequestration, Infantile hemangioma, Consumption coagulopathy, Portwine hemangioma, thrombocytopenia
Kasabach Meritt syndrome	Giant hemangioma
Quinine induced thrombocytopenia	Antibody mediated
Thrombocytopenia in newborn may be caused by	ABO Incompatibility
Thrombocytopenia caused by	Chlorpropamide, Quinine, Thiazide
Thrombocytopenia may be caused by	*Penicillin, abciximab*
An asymptomatic growth retarded infant is born by caesarean section for maternal hypertension. It is noted to have low platelet count. most likely diagnosis	Autoimmune thrombocytopenia
Autoimmune destruction of platelets	SLE,CLL,HIV
Megakaryocytic thrombocytopenia caused by	ITP,SLE,DIC

Palpable Purpura NOT occur in	Thrombocytopenia
Splenomegaly is NOT associated with	Essential thrombocythemia
Screening test for thrombocytopenia	Peripheral smear
Thrombocytopenia secondary to anticancer therapy treated by	Oprevelkin
Pseudothrombocytopenia	**Occurs if blood collected using EDTA (less calcium)**
Heparin induce thrombocytopenia presents with	*Thrombosis*
Treatment of heparin induced thrombocytopenia	Lepirudin
Ecarin clotting time drug monitoring for	*Lepirudin*
Treatment of HIT in patient with pulmonary embolism and hepatic failure	*Hirudin*
Treatment of HIT in patient with pulmonary embolism and renal failure	*Argatroban*
Treatment of Heparin Induced Thrombocytopenia	Direct Thrombin Inhibitor (argatroban, lepirudin)
Platelet transfusion is NOT indicated in	Immunogenic thrombocytopenia
MC cause of Neonatal Thrombocytopenic Purpura	Infection
Palpable purpura	Cryoglobulinuria, Henoch schonlein purpura, drug induced vasculitis
Purpura may rarely cause	Intususception
Hess test	Purpura

IDIOPATHIC THROMBOCYTOPENIC PURPURA

Cause of ITP	Antibodies to platelets
5 year old child, overnight petechial spots 2 weeks back, abdominal pain and no hepatosplenomegaly	Idiopathic thrombocytopenic purpura
ITP	Autoimmune, Increased megakaryocytes in marrow, IV immunoglobulins may be required, resolves spontaneously
ITP	Acute ITP often preceded by viral infection, chronic ITP common in females, acute ITP common in children
ITP is associated with	*Antiplatelet antibodies to GpIIb/IIIa, increased mean platelet volume, normal number of megakaryocytes in bone marrow, megakaryocytes show diminished budding*
Chronic ITP is common in	*Females*
Chronic ITP is due to	*Antibody production against platelets*
Immunoglobulin associated with Chronic ITP	IgG
MC cause of neonatal ITP	Infection
Abnormally prolonged in ITP	Bleeding time
ITP is NOT associated with	Splenomegaly
Bleeding into joint cavities NOT common in	ITP
Thrombotic event NOT seen in	ITP
False about chronic ITP	Splenomegaly
NOT true about chronic ITP	Spontaneous remission
Drug on choice in neonatal ITP	Gammaglobuin

Romiplostim is used in treatment of	*ITP*
MC Indication of Splenectomy in Elective Setting	Idiopathic Thrombocytopenic Purpura
Splenectomy is effective in	ITP though spleen is NOT enlarged
Platelet transfusion is NOT useful in	ITP

THROMBOTIC THROMBOCYTOPENIC PURPURA

Upshaw Schulman syndrome	**Congenital TTP**
Thrombotic thrombocytopenic purpura pentad	Microangiopathic hemolytic anemia, fever, disturbed neurologial function, renal failure, thrombocytopenia
TTP	**Protease activity is inhibited**
Tests for coagulation in TTP	Usually normal
NOT true about TTP	Grossly abnormal coagulation tests
NOT true about TTP	Renal failure is common
NOT seen in TTP	Hypertension
Treatment of choice for TTP	Plasma exchange therapy
NOT a treatment of TTP	Heparin, Platelet transfusion
Platelet transfusion are NOT indicated in	Thrombotic Thrombocytopenic purpura
Plasmapheresis used in treatment of	TTP
Plasmapheresis	Myasthenic crisis, Guillain barre syndrome, Polymyositis, TTP
Plasmapheresis is indicated in	Goodpasture syndrome, myasthenia
Plasmapheresis NOT indicated in	Cholinergic crisis

DISSEMINATED INTRAVASCULAR COAGULATION

Causes of DIC	Leukemia, massive transfusion, abruptio placenta, lymphoma, snake bite, adenocarcinoma prostate
NOT cause DIC during pregnancy	Diabetes mellitus
Disseminated Intravascular Coagulation won't occur in	Missed abortion
DIC	Increased PT, APTT, decreased fibrinogen, thrombocytopenia
DIC is most commonly seen in	Obstetric complications
DIC is seen in	Retained Placenta
Factors decreased in DIC	1,2,3,4
Bleeding in DIC closely related to	Low serum fibrinogen level
DIC commonly seen in	M3 AML
DIC differs from TTP that in DIC	Decreased coagulation factor levels
Clotting factor consumed during coagulation	Factor I fibrinogen
Finding in DIC	Increased FDP, prolonged PT, reduced platelets
Cutaneous manifestation of DIC	**Purpura fulminans**
Carcinoma associated with DIC	Stomach, pancreas, prostate
MC Site of Thrombi Formation in DIC	Brain (Brain>Heart>Lung>Kidney>Adrenal>Liver)
NOT abnormal in DIC	Clot solubility
DIC not occurs in	Prolonged pregnancy
NOT seen in DIC	Increased fibrinogen
DIC is NOT seen in	Fat embolism
Features of DIC does NOT include	Reduced PTT

Diagnosis of DIC by	Fibrin degradation products
Most specific investigation for DIC	Fibrinogen assays

ANTIPHOSPHOLIPID ANTIBODY SYNDROME

NOT a cause of congenital hypercoagulability	Anti phospholipid antibody syndrome
NOT an Inherited disorder	APLA
Isolated prolongation of aPTT with no history of bleeding	Lupus anticoagulant
35 year female, bilateral deep vein thrombosis, past history of recurrent fetal loss and one attack of pulmonary embolism. prolonged APTT	Anti phospholipid antibody syndrome
Lupus anticoagulants cause	Recurrent abortion, false postitive VDRL, arterial thrombosis, prolonged aPTT
Lupus anticoagulant	May present with isolated prolongation of APTT, present with recurrent abortion, may present with minimal clinical manifestations
APLA	Commonly presents with recurrent fetal loss, May cause pulmonary hypertension, Warfarin is given as treatment
APLA is associated with	Thrombocytopenia
Sapporo criteria for	*Anti phospholipid syndrome*
APLA is NOT associated with	Bleeding disorders
NOT a feature of APLA	Pancytopenia
NOT true about APLA	Single titre of anticardiolipin is diagnostic
NOT true about APLA	Thrombotic spells can be followed by severe life threatening hemorrhage
NOT true about Lupus anticoagulant	Increased PT
NOT associated with APLA	Thrombocytosis
NOT affected in lupus coagulant	Prothrombin time
NOT an indication of antiphospholipid antibody testing	Gestational diabetes
Anti β2-glycoprotein antibody	Antiphospholipid antibody Syndrome
Test to establish presence of lupus anticoagulant	Russel viper venom assay
35 year female normal PT, increased aPTT, operated for cholecystectomy, did NOT have bleeding episode	Anti viper venom assay
Recommended treatment for women with recurrent pregnancy loss associated with antiphospholipid syndrome	Aspirin and low molecular weight heparin
Used for monitoring low molecular weight heparin	*Factor Xa levels*

GENERAL FEATURES OF HYPERCOAGULABLE DISORDERS

Drug causing hypercoagulability	L asparaginase
MC Inherited cause for Hypercoagulability	Leiden Mutation
MC congenital cause of venous Thrombosis	Factor V Leiden
MC Hereditary blood coagulation disorder	Factor V Leiden
MC genetic cause of Thrombophilia	Factor V Leiden

Hypercoagulability due to defective factor V gene is	Leiden mutation
Inherited hypercoagulability that can lead to pulmonary thromboembolism	Activated protein C resistance (factor V Leiden mutation)
Hypercoagulation in nephrotic syndrome	Loss of antithrombin III
Coagulation defect associates with increased coagulation	Protein C resistance, Dysfibrinogenemia
Inherited coagulation disorders	Protein C deficiency, Protein S deficiency, Leiden factor mutation
Predisposing factor for venous thrombosis	AT III deficiency, Protein S deficiency, Protein C deficiency, Dysfibrinogenemia
Hyperviscosity is seen in	Cryoglobulinemia, multiple myeloma, lymphoma, waldenstorm's macroglobulinemia
Only condition causing both arterial & venous thrombosis	Hyperhomocystinemia
Predisposing arterial thrombosis	Homocystinemia
Risk factor for Thrombosis	High homocysteine
NOT a cause of congenital hypercoagulability	APLA
NOT a hypercoagulable state	Factor V leiden DEFICIENCY
NOT associated with thrombotic events	ITP
NOT predispose to thrombosis	Hypomagnesemia
NOT a cause for deep venous thrombosis	Diabetes mellitus

BUDD CHIARI SYNDROME

Commonest cause of Budd chiari syndrome	Thrombosis of hepatic vein, due to polycythemia rubra vera, myeloprolifetative syndromes, paroxysmal nocturnal hemoglobinuria
Budd Chiari syndrome	Hepatic vein thrombosis
Cause of Post hepatic Portal hypertension	Budd chiari syndrome
Spider web hepatic veins	*Budd Chiari syndrome*

HEMOLYTIC UREMIC SYNDROME

Picture resembling microthrombin in renal vasculature occurs in	Preeclampsia, Hemolytic uremic syndrome, Malignant hypertension
Causative organism of HUS	Shigella dysenteriae
Toxin associated with hemolytic uremic syndrome	**Shiga toxin by type I shigella dysenteriae**
Hemolytic uremic syndrome triad	Anemia, thrombocytopenia, renal failure
Hemolytic uremic syndrome	Uremia, hypofibrinogenemia, thrombocytopenia
Hemolytic Uremic Syndrome	Acute renal failure, Endothelial damage, Verotoxin induced injury
HUS	Often self limited, Fever is usually mild or absent, Usually follow hemorrhagic colitis
HUS	Infection may be transmitted by food, HUS is caused by serotoxin producing Escherichia coli, HUS is more common in children, HUS is commonly associated with hemorrhagic colitis
Shigella associated Hemolytic uremic syndrome is	Hyperkalemia, thrombocytopenia, renal microthrombi

associated with	
8 year old boy, diarrhea, decreased urine output, thrombocytopenia	Hemolytic uremic syndrome
MC toxin Implicated in Hemolytic Uraemic Syndrome	Shiga like toxin/Shigella Verotoxin
Most important toxin utilized in pathogenesis of hemolytic uremic syndrome	Serotoxin /verotoxin/shiga like toxin
NOT a feature of Hemolytic uremic syndrome	Splenomegaly
NOT true about HUS	Always fatal
NOT true about HUS	Decreased efficacy of BCG due to cross immunity
HUS NOT associated with	Vibrio cholera
NOT true about Hemolytic uremic syndrome	Positive Coomb's test
NOT a feature of hemolytic uremic syndrome	Neuropsychiatric manifestation
Finding NOT seen in Hemolytic Uraemic Syndrome	Thrombocytosis
Complement levels in hemolytic uremic syndrome	Normal
Seen in peripheral smear of hemolytic uremic syndrome	Schistocytes
Most indicative of hemolysis	Schistocytes
Coagulation profile in Hemolytic uremic syndrome	Normal
Kidney biopsy in child with Hemolytic uremic syndrome	Thrombotic microangiopathy

PLASMA CELL DISORDERS

GENERAL FEATURES OF MYELOMA

Smoldering myeloma	Plasma cells constitute 10-30% of total bone marrow cellularity
Smoldering myeloma	**No bony lesion, M protein > 30 g/dl**
Smoldering myeloma is associated with	Monoclonal gammapathy
Lichen myxedematous (papular mucinosus) is associated with	**Monoclonal gammapathy**
Church spine peak is associated with	**Monoclonal gammapathy**
<10% of bone marrow plasma cells, M component <3 g/dl	Monoclonal gammapathy of unknown significance
80 year old asymptomatic woman, monoclonal spike on serum electrophoresis. plasma cells 8%	Monoclonal gammapathy of unknown significance
False statement about MGUS	Bence Jones Proteinuria
Mu heavy chain disease is associated with	**CLL**
Franklin disease	Gamma heavy chain disease
Palatal edema is associated with	*Franklin disease*
Seligmam disease	**Alpha heavy chain disease**
Patient with IgD myeloma may present with	NO evident spike on serum electrophoresis
Plasmapheresis is done for	Myasthenia gravis, guillain barre syndrome, polymyosits
A pt has renal failure and bone pain. Serum M spike. 35% plasma cell. Diagnosis	Multiple myeloma
Church spire peaking	**Immunofixation usually in gamma globulin**

FEATURES OF MULTIPLE MYELOMA

Virus associated with Multiple Myeloma	HHV-8
Age predilection of multiple myeloma	More than 55 years of age and above
Commonest malignant bone tumor	Multiple myeloma
13q Deletion, 14q Translocation	Multiple Myeloma
Multiple myeloma is an abnormal aggregate of	Plasma cells
Multiple myeloma cells are dependent on	Il-6
MC Site of Multiple Myeloma	Vertebra
MC site of lytic lesion in multiple myeloma	Vertebral column
MC sign in multiple myeloma	**Bone pain**
MC cause of Death in Multiple Myeloma	Infection
Pseudohyponatremia	Multiple Myeloma
Multiple myeloma shows	AL type deposits
Moot Bodies	Multiple Myeloma
Russel bodies are seen in	Plasma cells
Russel's bodies are	Ageing plasma cells
Flame cells	Multiple Myeloma
Moth cells	*Multiple myeloma*
Bence Jones Proteinuria is seen in	Multiple myeloma
Bence jones proteins best described as	Kappa and lambda chain
Bence jones proteinuria may be seen in	Mu chain disease
Bence jones protein are derived from	Gamma globulins
Bence Jones protein in urine are due to presence of	Light chain of monoclonal immunoglobulins
Osteosclerotic multiple myeloma is commonly associated with	**Lambda chain**
Multiple myeloma	Visual disturbance, bleeding tendency, proteinuria
Maximum ESR seen in	Multiple myeloma
Renal biopsy of 56 year female, progressive renal failure past 3 years shows glomerular and vascular deposition of pink amorphous material. Apple green birefringence under polarized light after congo red staining. positive for lambda light chains	Multiple myeloma
Backache, recurrent infection, normal serum alkaline phosphatase, reversal of albumin globulin ratio	Multiple myeloma
Orbital mass, anemia, hypergammaglobulinemia	Multiple myeloma
Amyloidosis with macroglossia	Multiple myeloma
Lead to increased viscosity of blood	Multiple myeloma
Hyperglycemia and hypercalcemia is associated with	Multiple myeloma
POEMS syndrome is associated with	Multiple Myeloma
Increased viscosity of blood	Multiple myeloma
May be seen in multiple myeloma	Renal failure
Clinical feature of multiple myeloma	B cell prolymphocytic leukemia patients differ from those with B cell chronic lymphocytic leukemia
Metabolic abnormality in multiple myeloma	Hypercalcemia
Multiple myeloma	Monoclonal light chain may be detected in both serum and urine, Patients with IgD myeloma may present with no evidence of M spike on serum electrophoresis
Multiple myeloma is associated with	Sheats of atypical plasma cells
M band is absent in	Light chain disease

Multiple myeloma	Normal alkaline phosphatase, Monoclonal M band
Multiple myeloma	Monoclonal light chain may be detected in both serum and urine
Disappearing vertebra pedicle sign	*Multiple myeloma*
Least common feature of multiple myeloma	Hyperviscosity syndrome
Hyperviscosity syndrome is commonly associated with	*IgM multiple myeloma*
Poor prognostic factor in Multiple myeloma	Azotemia, increased LDH, chromosome 13 deletion, high M component production
Major criteria for multiple myeloma	Plasmocytoma on tissue biopsy, Bone marrow plasmacytosis > 30% of plasma cells, Monoclonal globin spike on serum electrophoresis or urine
M component in multiple myeloma	>3g/dl
Thymidine labeling index	>1%
NOT a major criteria for diagnosis for multiple myeloma	Lytic bone lesions
NOT a minor diagnostic criteria for multiple myeloma	Plasmocytoma on biopsy
Not a Multiple myeloma criteria	ANA
Raised serum alkaline phosphatase NOT seen in	Multiple myeloma
NOT seen in multiple myeloma	Polyclonal gammapathy, raised anion gap
NOT seen in multiple myeloma	Dystrophic calcification
NOT true about multiple myeloma	T(8,14) translocation
NOT true about myeloma	Protein cast in urine are made up of complete immunoglobin chains
NOT a feature of multiple myeloma	Elevated alkaline phosphatase
NOT a feature of multiple myeloma	*Proximal tubule defect*
International staging system for	*Multiple myeloma*
Multiple Punched Out Lesions	Multiple Myeloma
Lytic beveled lesions in skull X ray	Multiple myeloma
LEAST common cause of proximal lytic lesion of head of femur	Plasmocytoma
Components of POEM syndrome (Crow Fukase syndrome)	**Polyneuropathy (severe progressive sensorimotor), Organomegaly, Endocrinopathies, M protein, Skin changes**
Demyelination is associated with	**POEM syndrome**
POEMS syndrome does NOT include	Ovarian dysgenesis
NOT a component of POEMS syndrome	Multiple osteolytic lesions

MANAGEMENT OF MULTIPLE MYELOMA

Tumor marker for multiple myeloma	Immunoglobulins
Feature of multiple myeloma	*Raised serum calcium*
The investigation, plasma viscosity is typically reserved for	*Multiple myeloma*
Beta 2 microglobulin is marker of prognosis for	Multiple myeloma
Beta 2 microglobulin is derived from	B cells
Multiple myeloma is diagnosed by	Bence jones proteinuria, Bone marrow plasma cells > 30%, Lytic bone lesions
Multiple Myeloma is diagnosed by	>10% plasmocytoma
Bone scan on patient of multiple myeloma shows	Cold spots
A patient with back pain. Elevated ESR. X ray shows lytic	Serum electrophoresis

multiple punched out lesions. Most important investigation	
Confirmatory test for myeloma	Serum electrophoresis
Investigation in multiple punched out lytic lesion in skull	Protein electrophoresis
Least useful investigation in multiple myeloma	Bone scan
Treatment of Multiple myeloma	Melphalan
NOT used for management of multiple myeloma	Hydroxyurea

DRUGS ACTING ON BLOOD

GENERAL FEATURES OF DRUGS ACTING ON BLOOD

Folic acid	Pteroyl glutamic acid, Useful in carriage of one carbon atom moiety, Tetrahydrofolate is active form
Anticoagulant for performing coagulation studies	Trisodium citrate
Anticoagulant for Prothrombin testing	3.2 % Trisodium citrate
Anticoagulant for blood glucose estimation while working in PHC	Potassium oxalate + Sodium fluoride
Use of anticoagulants in patients with lower limb surgeries	*Delay placement of spinal or epidural catheters 12 hours after last dose of LMWH, LMWH should be give no sooner than 2 hours after removal of epidural catheter, Neuraxial catheters should be put and removed only if INR < 1.5, warfarin to be discontinued at least 72 hours before surgery,*
Blood collection tube with purple top	**EDTA**
Blood collection tube with green top	**Heparin**
Blood collection tube with blue top	**Sodium citrate**
Filgastrim	G-CSF
Filgastrim used in treatment of	Neutropenia
Orally activating direct thrombin inhibitor	Ximelgatron
Direct acting thrombin inhibitors	Argatroban, ximelgatron, bivalrudin
Oral direct thrombin inhibitors	*Ximelgatron, dabigatron*
Parenteral bivalent direct thrombin inhibitors	*Hirudin, lepirudin, bivalirudin*
Parenteral monovalent direct thrombin inhibitors	*Argotroban, megaltaron*
Parenteral thrombin inhibitor associated with shortest half life	*Bivalirubin*
Which is NOT a Parenteral Direct Thrombin Inhibitor	Dabigatron
Proved antithrombotic property	Dextran 40
Disadvantage of dextran	Interference with blood group matching
NOT used for thromboprophylaxis	Antithrombin III
NOT an anticoagulant	Phytonadione
Oral anticoagulants are monitored by	PT
Plasma expanders used in	Severe trauma

Gp IIb/IIIa antagonist	Abciximab
Abciximab	**No renal clearance**
Apixaban	Directly inhibits factor Xa, thereby interfering with the conversion of prothrombin to thrombin and preventing formation of cross-linked fibrin clots
Activated protein C therapeutically used in	Sepsis
Number of saccharide units in fondaparinux	**5**
Aggrenox	*LMWH + dipyridamole*
Fondaparinux	*Direct Xa inhibitor*
Newer direct Xa inhibitor	*Apixaban, rivaroxaban*

HEPARIN

Heparin is structurally	Heteropolysaccharide
Heparin acts via activation of	Antithrombin II
Low molecular weight heparin inhibit	Xa
NOT true about heparin	Weakest acid in living beings
Heparin does NOT cause	Factor V inhibition
Heparin therapy monitored with intermittent estimation of	PTT
Anticoagulant of choice in pregnancy	Heparin
LMWH	Absorbed more uniformly when given subcutaneously
Causes hyperkalemia in a patient with diabetes and estimated GFR within normal reference range	*LMWH*
A diabetic female on INH and rifampicin for TB developed DVT, she was started on warfarin, PT is NOT raised and next step should be	Use LMW heparin
Side effect of heparin	Hyperkalemia
NOT a side effect of heparin	Skin necrosis, Hypokalemia
Drug used for heparin overdose	Protamine sulphate
Hemorrhage secondary to heparin administration corrected by	Whole blood

WARFARIN

Half life of warfarin	36 hours
Warfarin is not used to prevent blood coagulation in blood collection centres because	*Anticoagulant effect of warfarin occurs only in vivo*
In a patient with prosthetic valve, warfarin should be replaced by pregnancy after	36 weeks
NOT true about warfarin	Dose increased in liver disease
In a warfarin treated patient skin necrosis is found in	Protein C deficiency
NOT a cause of therapeutic failure of warfarin	Rifampicin
NOT true about warfarin skin necrosis	Most common sites are toes and tips of fingers
Effect of coumarin therapy reversed by	Vitamin K

Urgent reversal of warfarin induced bleeding	Fresh frozen plasma
Warfarin skin necrosis	**After 3 – 10 days of treatment, treated with vitamin K, protein C concentrate**

ANTIPLATELET DRUGS

Platelet aggregation is inhibited by	Aspirin, clopidogrel, dipyrimidole
Aspirin prolongs bleeding by inhibiting synthesis of	Thromboxane A2
In low dose aspirin acts on	Cycloxygenase
Aspirin cause	Platelet dysfunction
Aspirin is contraindicated in patient on treatment with	Warfarin
NOT true about aspirin	Duration of action is primarily related to pharmacokinetic clearance of drug from the body
Ticlopidine	Delayed onset of action, long duration of action, alternate to aspirin in patients with cerebrovascular disease
Superior antiplatelet drug	Ticlopidine
Ticlopidine acts on	ADP receptor, Onset is rapid and duration is longer
Clopidogrel acts by	Inhibiting ADP induced platelet aggregation
Cytochrome involved in decrease in efficacy of clopidogrel by PPI	Cytochrome 2C
Newer antiplatelet drugs	**Cangrelor, Ticagrelor (directly acting reversible PY12 antagonists)**
Ticagrelor	**Reversible inhibitor of P2Y12**
Tirofiban	Antiplatelet drug
Mechanism of action of vorapaxar	*Platelet activated receptor 1 inhibitor*
Dazoxiben	*Thrombaxane synthase inhibitor*
Saltroban, losartan	*Thromboxane A2 receptor blocker*
Picotamide	*Thromboxane synthase inhibitor, thromboxane A2 receptor blocker*
Anagrelide	*Platelet maturation inhibitor*
NOT an antiplatelet	Warfarin

THROMBOLYTICS

Treatment of choice in a patient of acute pulmonary embolism with right ventricular hypokinesia and a compromised cardiac output but normal blood pressure	Thrombolytic agent
Absolute contraindication to thrombolytic therapy	History of hemorrhagic stroke in past 1 year
NOT a contraindication to thrombolytic therapy	Hypotension
NOT a contraindication of thrombolytic therapy	SVT
Thrombolytic agent that can cause plasmin activation	Streptokinase
Alteplase differs from streptokinase as it	Activates plasminogen bind to fibrin
Plasminogen activator can be given as bolus dose in patients with acute MI	Reteplase
Epsilon amino caproic acid is used to reduce bleeding due to	Hyperplasminemia
EACA is used in treatment of adverse effects caused by	streptokinase

Inhibitor of fibrinolysis	Tranexemic acid
Traenexemic acid reduces blood loss by	50%

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